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THE SURGERY OF ACUTE CHOLECYSTITIS *

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The present status of surgery in acute cholecystitis is a culmination of three decades of profound controversy. In 1923, Walton [80] first urged early surgical treatment, rather than delay, in all cases of acute cholecystitis. He cited the remarkable, rapid postoperative convalescence. The following year, Bland-Sutton [10] stated all cases should be operated upon immediately, saying it was "reprehensible" not to remove the acute gall bladder. Again, in 1924, Kirschner [45] advocated early operation because he believed surgery was easier, and that the patient was indeed more willing to be operated upon during the attack. Kirschner's philosophy may well be compared with the long prevailing attitude in strangulated hernia and acute appendicitis. In the United States, George Crile, quoted by Bruggeman [16] in 1928, stated he was greatly impressed by his excellent results with cholecystectomy in acute disease. In the same paper Leriche and Cotte also urged that cholecystectomy should be done immediately, Bruggeman asserting that French surgeons generally advise immediate operation.

During the same period the hue and cry of the opposition—urging delay—voiced by Enderlin and Holtz,[27] in Germany, Lyons and Judd [51] in this country, followed by Deaver [21] and Evarts Graham,[36] did not deter those who had been convinced by their good results. The imposing array of opposition contended that most acute cholecystides subsided without complications and that perforation was rare. If one awaited abatement of the acute process, surgery could be undertaken from three to six weeks after recovery, with a lower mortality rate.

There soon were published, in this country, a number of papers, Alexander,[1] Miller,[58] H. F. Graham and Waters,[37] Heuer,[40] Mentzer,[56] Lobingier,[50] Mentzer,[57] Zinninger,[85] Judd and Phillips,[44] Smith,[71] Stone and Owings [72] to substantiate the earlier advocates of immediate and early cholecystectomy. Alexander's series revealed that in 20 patients who were carefully observed and treated medically, perforation had occurred. There was a mortality of 50 per cent. Mentzer (1932) presented 8 patients, undergoing medical therapy, all of whom died. Thirty-one patients, also undergoing prolonged medical treatment, advanced to perforation or gangrene before surgery was performed. The mortality in this group was 33 per cent. Zinninger's elaborate study showed that in his patients, operated upon within forty-eight hours after onset, there was no mortality. Those operated upon within two to five days, the mortality was 6.6 per cent. In those acute cholecystides operated upon after five days the mortality rate increased to 25 per cent. Probably the best of the early papers was

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George Heuer's review in 1937 [41] in which he presented 153 cases of acute cholecystitis operated upon by him. Of this number, 65 per cent were operated upon within twenty-four hours, 35 per cent within seventy-two hours. In his entire series there were 5 deaths, a mortality rate of 3.2 per cent. In this series 40 of the 153 gall bladders showed areas of gangrene, and in 16 of these the gall bladder was perforated on admission. His critical analysis of his own series reveals there were 137 cases of acute cholecystitis without perforation with 3 postoperative deaths—a mortality rate of 2.1 per cent. In the 16 cases with perforation there were 2 deaths—a mortality rate of 12.5 per cent.

The recent surgical literature, 1940 to 1955, has produced a great number of excellent articles which sustain the pioneers' efforts to establish early cholecystectomy as the operation of choice in acute cholecystitis, especially Glenn,[31-35] Dunphy, Ross et al.,[25, 67, 68] Wallace and Allen,[78] Clifford,[19] Eliason and Stevens,[26] Lester [47] and Best.[8] An excellent dissertation on acute noncalculous cholecystitis and its pathology is found in Wolfson and Rothenberg's paper.[84] The most enlightening and substantiating review of the pathology in acute cholecystitis was presented in a clinical paper on the subject by Touroff.[76] Recently (1952), an exhaustive article on the subject of timing in surgery of acute cholecystitis was published by DeCamp, Ochsner et al.,[22] substantiating Ross, Boggs and Dunphy.[68] In 1953, Frank and Orr [30] again emphasized the philosophy of the two latter groups, stating that "operation is indicated *as soon as the patient can be prepared,* providing the diagnosis is reasonably well established and there are no serious contraindications to surgery". Pines and Rabinovitch [62] (1954) review 1480 patients operated upon for acute cholecystitis with 6.5 per cent mortality in uncomplicated cases. Where perforation has occurred the overall mortality rate was 17.7 per cent. This paper presents an excellent statistical summary of the literature on perforations of the gall bladder. Their large series of patients (1480) were gathered from the records of the Brooklyn Jewish Hospital from 1932 to 1952—a large general hospital with many resident staff operators as well as those of the active attending surgical staff. They conclude that "the *optimum time for surgery* is often missed because patients are not hospitalized nor operated upon until many days after the onset of symptoms—in the authors' series, *more* than ten days".

It is most significant at this time to note that the internist has been following his patient to the operating room and has drawn his own conclusions, as he had done in acute appendicitis with its controversies in the 1890 to 1910 period, and in strangulated hernia in the 1870 to 1890 period. J. Edward Berk,[7] Associate Professor of Medicine, Wayne University, Detroit writes—"If during the first 24 or 36 hours, the temperature and pulse rate continue to mount, the local signs of inflammation increase, and the leukocyte count shows progressive elevation, operation is no longer delayed. Similarly, if after 36 to 48 hours, the temperature has not significantly lowered, the pulse rate remains rapid, the abdominal signs are essentially unchanged, the white blood count is still elevated, and the patient generally appears unimproved, operation is no longer delayed. The operative procedure of choice is cholecystectomy, with or without common duct exploration as indicated by the history and operative findings. When the findings at the time the patient is initially seen suggest perforation or imminent perforation, surgical intervention is elected with the least possible delay".

PATHOGENESIS

Acute cholecystitis occurs with and without the presence of gall stones. Acute noncalculous cholecystitis is often the result of the invasion of the wall of the gall bladder through the lymphatics of the liver (Graham et al [36]), or hematogenous bacterial infection (Rosenow,[66] Rehfuss and Nelson[64]). Boyd [12] states that ascend-

ing infection from the duodenum does not occur. Pancreatic reflux, with its necrotizing effect, has been reported in cases of acute noncalculous cholecystitis by Colp and Doubilet[20] and Bisgard and Baker.[9] An embolus or an atheromatous intima of the cystic artery, or torsion of the cystic duct may produce gangrene of the gall bladder. Wolfson and Rothenberg found 31 cases of noncalculous cholecystitis in a series of 279 patients with acute cholecystitis—a percentage of 8.2. Allen Whipple[82] in a series of 160 patients found 30 without stones, or 18.7 per cent. Infection produces inflammatory swelling of the lining of the gall bladder and the cystic duct. This swelling produces obstruction at the ampulla or duct, and the ensuing pathological process is similar to that which produces the same sequence of events in stone impaction of the ampulla or duct.

Strohl and Diffenbaugh[73] reported 118 cases of acute cholecystitis in which 85.6 per cent contained stones. Numerous reports to date indicate that the cause of acute cholecystitis is due to obstruction of the ampulla or cystic duct in at least 95 per cent of cases.

The most common sequence of events follows when small stones may become impacted in the cystic duct, or a larger one in the ampulla, obstructing the outflow of bile (Figures 1 and 2). It should be noted that there are four vascular systems, or their components, involved in the mechanics of obstruction in the acute gall

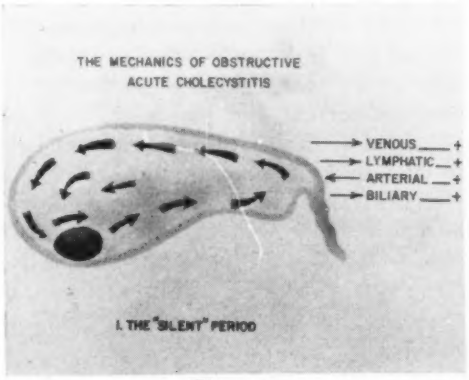

Figure 1

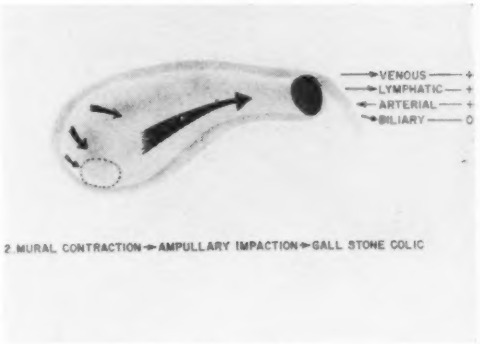

Figure 2

bladder: (1) the biliary components, the ampulla of the gall bladder and the cystic duct and the flow of bile to and fro; (2) the arterial component, the cystic artery—acts as it were a true end-artery despite the numerous gall bladder fossa capillaries, (The artery is usually single but may be multiple); (3) the venous components, the cystic vein which empties into the right branch of the portal vein and minute radicals which may empty into the liver; (4) the lymphatic components, the main vessels of which empty into the nodes at the neck of the gall bladder. (Figure 1).

With the impaction of the stone in the ampulla (surgically, Hartmann's pouch) or cystic duct the outflow of bile ceases—thus setting up, in a very small and restricted area, (about the triangle of Calot, especially) a progressive compromising of the circulation of blood and lymph in the entire gall bladder wall. All major portals of egress and ingress are confined to an area of 3 cm. generally about the ampulla. With the development of spasm and edema about the wall of the ampulla and the cystic duct, the lymphatic return and the venous outflow are first to become slowed and may eventually cease (Figure 3). As seen so frequently at surgery of the acute gall bladder the cystic artery seems to be the villain of the play. It becomes distended, larger, bounding, and is frequently seen to pulsate. It is not infrequently as large as the radial artery in the fulminating picture. Since it seems to act not unlike an end-artery, the blood continues to

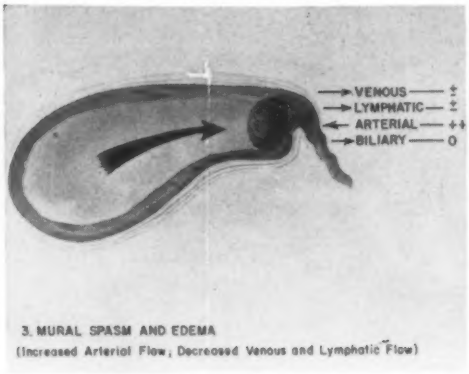

Figure 3

pour into capillaries of the viscus; edema, subserous, mural, as well as submucosal, is progressive due to lymph stasis; venous congestion due to venous block, with its concomitant hypoxia, plus inflammatory changes in the mucosa, submucosa, the Pokitansky-Aschoff sinuses; destruction of capillary walls with its diapedesis of red cells as well as frank hemorrhage into the interstices; focal areas of necrosis begin to appear in the wall. It could be explained as a problem in hydrostatics, with the capillary bed and its delicate walls, the site of initial disruption. The earliest areas of ischemic necrosis are more frequent in the fundus, farthest from the ampulla, where the blood supply has been further compromised by greatly increased intracystic tension and pressure. It is thinnest in this area also. Ruptures are most frequent here. However, blotches of gangrene may be found in various stages of progress throughout the entire wall (Figure 4).

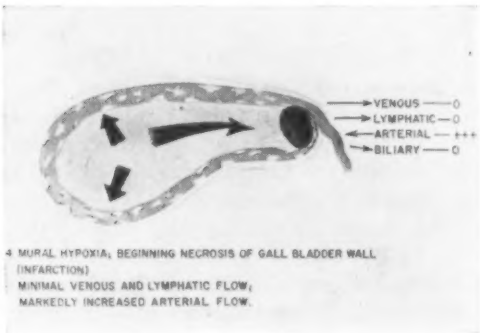

Figure 4

Figures 1 through 4 are representative of culminating ideas gathered from the surgical and pathological literature, inspired mainly by Lobingier,[50] Homans,[42] Denton,[23] Andrews,[2] Hallendorf, Dockerty and Waugh[38] and others. The entire sequence can be likened to that produced by the torsion of an ovarian cyst pedicle; acute obstructive appendicitis (Van Zwalenberg,[77] McCallig[52]); volvulus of the sigmoid or small bowel, or as seen rarely in torsion of the mesentery of a ptotic gall bladder. How many acute cholecystitides have resolved, completely or partially, due to abnormal and/or accessory veins, or accessory arterial systems, can only be conjectured. The unusual number of abnormalities in the "cysto-hepatic angle" (Moosman and Coller,[59] Browne,[15] Flint,[28, 29] Brewer[14] et al.) should render unlikely only one mechanical process as all prevailing. The rich network of fine lymphatic radicles between the liver bed and the gall bladder wall may be considered as another good "escape hatch". The question of bacterial inflammatory complications of the picture cannot be dismissed as an entirely negligible factor. Various pathogens have been cultured from the fundic bile, as well as the wall, by many. The main groups are streptococci, staphylococci, the anaerobes (especially *C. Welchii*) and various members of the *E. Coli* family. (Rosenow,[66] Magner and Hutcheson,[55] Graham, et al.[36]). Andrews[2,3] believes that bacteria play only a minor role in the great majority of the acute cholecystides. McKibbon and McDonald[53] support his view, and point out that polymorphonuclear leukocytes are frequently present in one or more layers of the gall bladder wall of apparently normal gall bladders. Andrews, Schoenheimer and Hrdina[3] ligated the cystic duct and the common duct producing biliary stasis, showing that infection of the gall bladder wall by anaerobic organisms, which seemed to pass from the liver to the gall bladder by direct extension, occurred in their experiments. The anaerobic organisms found in many cases of cholecystitis correspond to the normal flora of

the liver, which is intestinal in origin. A review of *E. Coli* septicemia due to acute cholecystitis was presented by Lipshutz and Kaplan[49] in 1941. They report 12 cases, 3 personal and 9 from the literature to 1941.

In comparing inflammation in the gall bladder with inflammation in other hollow abdominal organs, such as the appendix or the urinary bladder, one finds striking differences. In the appendix, and in the urinary bladder, there is a close correlation between the pathological changes and the clinical symptoms, and bacteria are present in enormous numbers. In acute cholecystitis the histological picture is rarely one of acute suppuration—and one finds very little difference, as a rule, between the bacterial count of acute and chronic cholecystides. Moreover, the clinical picture in early acute cholecystitis and late, so-called subsided acute cholecystitis, rarely corresponds to the pathological picture. As Touroff so vividly pointed out in his series, 99 per cent of the patients operated upon after symptoms were minimal or absent, still showed acute cholecystitis histologically for periods as long as three years after the acute clinical episode had subsided. Many authors have commented on similar observations. It is not too rare to find the pathologist's report on a so-called elective operation upon chronic cholecystitis with cholelithiasis revealing "acute diffuse cholecystitis with cholelithiasis".

THE DIAGNOSTIC PICTURE

Most patients with acute cholecystitis fall within the 30 to 60 year old group. Acute cholecystitis in childhood is relatively rare, but in 1938, Potter[63] reported 432 cases from the literature, of biliary disease in the young (under 15 years of age), two-thirds of which were acute cholecystitis and chronic cholecystitis with cholelithiasis.

A history of previous attacks is often noted, in the majority of cases, but usually less severe than the presenting attack. A sudden onset of severe localized pain in the right upper quadrant usually initiates the attack. However, the onset may be gradually noted, with epigastric or periumbilical pain, soon localizing over Mayo-Robson's point. Radiation of pain may vary—to the right costo-vertebral angle, the right scapula or right interscapular space (splanchnic nerve distribution) or to the right shoulder. Anorexia is an early symptom, as is nausea. Most patients vomit—and vomiting is often incessant. Chills are a danger sign—indicating early gangrene or perforation. In older patients a clear cut history is not the rule, but the exception. Patients in the decades past 50 years present the most trying problems in diagnosis. This is becoming more and more important. In a series of 586 patients Glenn and Moore[32] reported 35 per cent over 50 years of age.

Physical examination usually reveals an acutely ill and apprehensive patient. Fever is usually low grade though many patients are afebrile. Fever cannot be relied upon to correlate the extent of the underlying disease or its pathological state (Bockus[11]). The pulse usually parallels the temperature rise. Precipitous rise, to 103-105°, means gangrene and/or perforation has occurred.

Tenderness and increased muscle tone in the right upper quadrant is almost always present. Mass is found in 15 to 25 per cent of patients. Even the rounded fundus of the enlarged gall bladder may be palpated or outlined by delicate percussion. The mass usually consists, however, of an intraperitoneal walling off process—the stomach and duodenum, transverse colon, and hepatic flexure, and omentum, mainly.

Jaundice, as a presenting sign indicates a serious complication, found in 10 to 20 per cent of statistical studies. Spreading inflammatory edema extending to the common bile duct or common hepatic duct, or stones or gravel in the common duct are the most common causes. Greatly enlarged lymph nodes along the common duct have been found to be the sole cause upon rare occasions.

The leukocyte count is often indicative of the severity of the disease process, especially under 50 years of age. Most

counts fall between 10 and 15,000. Past 50 years of age, white counts, like the temperature rise, are often misleading. Normal counts often go along with normal or subnormal temperature in the older age groups. Counts higher than 15,000 with pronounced shifts to the left often mean impending or actual gangrene and/or perforation.

The urine may show bile, and, on occasion, sugar in nondiabetics, because of pancreatic involvement. Proteinuria, granular and hyaline casts amongst the formed elements are not unusual, especially with marked fever rises.

The serum bilirubin should be determined, whenever possible, preoperatively, to detect not only subclinical jaundice but also, in jaundiced patients to follow postoperative progress.

The color of the stools should be noted for acute biliary obstruction, complete or partial.

The color of the vomitus, particularly of prolonged vomiting, is an indicator of the presence or absence of duodenal bile.

A good routine procedure to be recommended is a scout film of the abdomen (K.U.B.). Not only may it show radiopaque stones or calcific gall bladder walls but also gas in the gall bladder fundus or biliary tree. Bell, Brown and Lenhardt [6] report 27 clinically proven cases of pneumocholecystitis up to 1953. An amazing 46 per cent in 121 cases of acute cholecystitis, proven at surgery, revealed a positive x-ray for radiopaque stones preoperatively, was reported by Smith [70] from the Presbyterian Hospital, New York (1945).

DIFFERENTIAL DIAGNOSIS

Fortunately, the diagnosis of acute cholecystitis in the hands of most practitioners of medicine and surgery has not proved to be too great a stumbling block. The usual clear cut history, so often laden with typical though mild acute attacks of pain in the right upper quadrant, selective food disturbances, pyrosis, colic and classical reflex pain distributions, outlines the diagnosis before the patient is examined. The well localized pain, muscle spasm, and marked resistance plus exquisite tenderness in the region of Mayo-Robson's point aid in arriving at a diagnosis within six to twenty-four hours after onset.

Though there are pitfalls, such as ruptured peptic ulcer, acute pancreatitis, late perforative appendicitis, right sided diaphragmatic pleurisy, lobar pneumonia, hiatus hernia, they are rather infrequent in large series. High intestinal obstruction, though rare, may mimic acute cholecystitis. Linder and Morse [48] present an extensive differential picture, quoted almost in toto by Thorek,[75] concerning acute pancreatitis.

The most intriguing of errors, from the viewpoint of the experienced internist and the surgeon is often found in the relationship between coronary artery disease and the affected gall bladder. The literature is abundant on the subject, since Babcock [4] reported 13 cases in 1909. As long ago as 1878, de Mussy [60] called attention to this frequently found association. That gall bladder disease could cause cardiac signs and symptoms, producing anginal pain and even toxic effects, along with inverted or flat T-waves in the electrocardiogram, have been shown by Breitweiser,[13] Cady et al.[17] and Weiss and Hamilton.[81] Walters and Master [79] believe that following cholecystectomy for acute cholecystitis, or for gall stones, the cardiac condition may be improved. The acute anginal pain and other cardiac symptoms may disappear, as may also the abnormal electrocardiographic changes. However the underlying disease of the coronaries is not affected by such surgical procedures. They state that "since both conditions often occur in the same age group, and since both may have a common etiologic basis (vagovagal reflex, atherosclerosis) their frequent association is probably coincidental".

To the surgeon, as well as the physician, it is extremely important to differentiate between acute cholecystitis and acute coronary occlusion, for surgery could prove disastrous. Wolferth [83] shows that there should be little difficulty in distinguishing between the pain of gall bladder colic and

angina pectoris if a careful history has been taken. As is well known, epigastric pain may occur with angina pectoris; invariably, however, anginal pain radiates under the sternum and often to the shoulders and down the left arm. It does not tend to radiate to the subscapular region. It is usually of a characteristic pressing or clutching quality, and is hardly ever centered in the right upper quadrant of the abdomen. With actual coronary occlusion or infarction the patient appears more critically ill,— pallor, even cyanosis, difficulty in breathing, with substernal pain are much more marked than abdominal pain. Fever, leukocytosis, rapid pulse are soon manifest. Because of the frequent association of the two diseases due deliberation and caution are paramount in deciding the issue of surgery or watchful waiting.

THE SEQUELAE OF AMPULLARY OR CYSTIC DUCT OBSTRUCTION

The passage of a relatively small calculus through the tubular structure of the ureter causes much pain and spasm. That there is an inflammatory reaction accompanying the passage is well manifested by the outpouring of red and white cells. The inflammatory edema and spasm in the cystic duct and its passing stone, with the additional obstructive hurdle of the valves of Heister, presents a similar pathological picture. With a large stone impacted in the ampulla or in Hartmann's pouch the felony is compounded similarly, though egress is not the rule. The same intracystic pressure which lodges the stone in the ampulla or pouch may act to dislodge the impaction, thus relieving the obstruction to the neck and cystic duct. Thus it may be assumed that the so-called mild or even more severe attacks of gall stone colic are accompanied by varying degrees of inflammatory process. It may also be assumed that the inflammation may not leave any scars of consequence after relief of impaction or passage. (Figure 5) Here one thinks again of Kirschner's philosophy—rarely does a patient have but one attack of gall stone colic; that eventually the attacks will become more severe; that

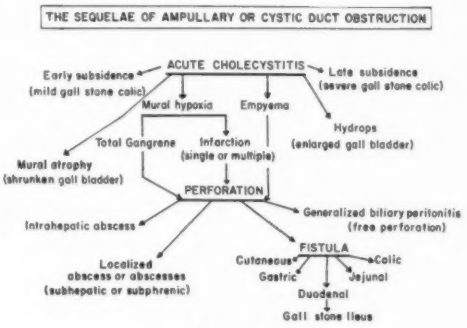

Figure 5

the optimum time to convince a patient surgery is imperative presents itself during an acute bout with stone. Hertzler[39] in his own inimitable fashion of "The Horse and Buggy Doctor" presents the picture of gall stone colic in the following paragraph taken verbatim from one of his many fine monographs: "When the gall bladder finds itself unable to expel its contents due to the presence of a stone in the duct it gets itself all het up, and this is expressed in terms of spasm.... Aside from the discomfort caused by the preliminary colic, the essential factor is inflammation of the gall bladder and its environs. Pain itself never killed anybody. As far as the welfare of the patient is concerned, the disease resolves itself into a study of inflammation. An acute attack after the first few hours is expressive of acute inflammation".

Figure 5 is a schematic outline of the consequent courses following an attack of acute cholecystitis.

The shrunken gall bladder (mural atrophy) may follow any attack or repeated attacks of acute cholecystitis. Stones are often embedded in its walls—in the Aschoff-Rokitansky sinuses or even in the ducts of Luschka. It is often an accompaniment of common duct stone. Though true empyema is rare it may lead to perforation with dire consequences. The large gall bladder seen in hydrops is an inert mucus or serous fluid filled sac, often extending in its immensity, and sometimes felt as a tumor, in the right iliac fossa.

As seen in Figure 4 the dangers of

hypoxia, with its resultant mural infarctions or total gangrene lead to perforation. Diffenbaugh, Sarver and Strohl[24] of St. Luke's Hospital, Chicago reported perforation in 11.9 per cent in a collected series of 5272 cases of acute cholecystitis. The mortality in the perforative series was 23.8 per cent. One is reminded of another disease with dire sequences, in crude comparison. At the John Gaston Hospital, the University of Tennessee teaching hospital 689 cases of acute appendicitis with perforation were reviewed. Of 3687, 16.7 per cent cases of acute appendicitis were perforated upon admission. The mortality for the series from 1945 to 1951 (364) was 6.6 per cent.[74] The indictment against delay in surgery of acute cholecystitis is the obvious parallel.

Free perforation, with its ensuing fatal generalized biliary peritonitis, fortunately is a rare complication. Intrahepatic abscess, due to perforation into the liver substance, is an uncommon occurrence. It may be single or multiple—the abscess may or may not contain extruded stones.

The protective mechanism of taxis is the most common reaction of the neighboring organs in the right upper abdomen. The transverse mesocolon, the transverse colon and hepatic flexure plus the omentum, and often the duodenum and stomach are found enmeshed in a more or less firm fibrinous exudate, walling off the impending or actual perforation. There, between the above mentioned organs and the inferior liver surface, one may find localized abscess or abscesses, extruded stones and bile. Areas of partial or complete mural ischemia and/or necrosis are often in evidence. Fistula results from contiguous necrosis of the walling off organs, the most common of which is the cholecysto-duodenal fistula. From cholecysto-duodenal fistula large stones are extruded that may produce gall stone ileus in the narrowing portion of the terminal ileum. The mortality rate is high in gall stone ileus as diagnosis is difficult and perforation of the ileum often is reported (McLaughlin and Raines[54]). These large stones, rarely pass through the cystic duct, but through the first or second portion of the duodenum, via an acute cholecystoduodenal fistula. If gall stones are vomited they are usually pathognomonic of cholecysto-gastric fistula. Stones passed in the feces, if large, are indicative of cholecysto-colic fistula. The scout films of the abdomen, in the majority of these fistulae, reveal gas in the biliary tree. McLaughlin and Raines report a series of 366 cases of gall stone ileus.

Cholecystectomy for acute cholecystitis was first performed by the author in 1939, stimulated primarily by Heuer's convictions and then translated into action by the conclusions of Carter, Greene and Twiss (1939[18]). The results were encouraging from the beginning. The report of Wallace and Allen, in 1941, confirmed not only the imperative need for early operation after proper preparation, but also revealed an important factor previously unemphasized. In 415 cases of acute cholecystitis they reported gangrene present in 125 cases—29.4 per cent. Perforation was found in 64 cases of the 125 gangrenous gall bladders, or an overall percentage of 15.4 in the 415 reported. They found that gangrene with perforation is unusual before the sixth day of onset of symptoms, and that operation performed within four days of the onset of symptoms is relatively safe if proper preoperative preparation is meticulously deliberated.

During the past few years surgery has been extended in so-called "delayed acute cholecystitis" as recommended by Ross, Boggs and Dunphy and DeCamp, Ochsner, et al.,[22] and long ago suggested by Wallace and Allen. The "proper timing" of surgery in acute cholecystitis, they believe, is not confined entirely to the early three or four day period. The resolution of the inflammatory process is extremely slow and incomplete regardless of the clinical course of the patient. Touroff's pathological studies record this fact most convincingly. After a short deliberative period, devoted to restoring fluid and electrolyte balance, the institution of antibiotic

therapy, the proper leveling of the hematocrit, the establishment of the status of the cardiovascular system and a daily urinary output of better than a liter, "there is nothing to gain by delaying surgery". The inflammatory process may persist for months or even years after the clinical symptoms subside. They conclude, as their experiences have shown, that the concept of an arbitrary period during which surgery for acute cholecystitis is to be avoided, should be abandoned. Contrariwise, with the patient's condition going from bad to worse, a life-saving cholecystectomy, under intercostal anesthesia (Bartlett[5]) may be the only life-saving measure.

THE MANAGEMENT OF TECHNICAL PROBLEMS

The dangers in surgery of acute cholecystitis parallel the pitfalls encountered in chronic biliary disease. In the early acute diffuse cholecystitis, the operation is often easier in execution, due to the easy dissection afforded by demarcation of structures by edema. But the "easy case" rarely presents difficulties or anomalous vessels. The avoidance of these dangers and pitfalls may be summarized in a "Duodecalogue" which might aid in guiding one to Jericho without struggling through the Wilderness. They contain the admonitions and friendly reproofs of Lahey, Coller, Ochsner, Sanders and other excellent technicians too numerous to mention.

1. Do not attempt the operation if you are an occasional operator. As a general practitioner you have enough of headaches and worries without adding another.

2. Do not enter the operating room before you have secured an experienced and able surgical assistant.

3. Do not enter the operating room before you have secured a capable anesthesiologist or nurse anesthetist. Pentobarbital, curare or curare-like drugs require split-second attentions. High spinal analgesia often requires additional intravenous or inhalation complement.

4. Do not make your incision if the surgical lighting system is defective or inadequate for deep focus.

5. Do not operate without your best surgical spectacles. Lahey, with his spectacles *and* his Loupe would impress any bystander with the critical importance of seeing what you are doing in the deep recesses of the abdomen.

6. Do not expect to find "normal anatomy" in every triangle of Calot. This holds true in routine cholecystectomy as well.

7. Do not anticipate the degree or the extent of the pathological picture. See to it that the instrument table is well stocked for any complicating procedure. Remember that cholecystostomy may be the procedure of choice; free perforation; acute pancreatitis; advanced hepatic disease; friable duodenitis; the frozen common duct from previous attacks; poor identification of structures due to progressive inflammation.

8. Do not grasp blindly in a pool of blood with any instrument smaller than your hand. Place your index finger in the foramen of Winslow; raise the hepatic artery gently; compress it with your index finger and thumb. Then use the aspirator.

9. Do not dissect, clamp or tie the cystic duct before the cystic artery has been ligated and cut free. This maneuver will obviate the pool of blood "accident". Traction on the ampulla or Hartmann's pouch, when the cystic duct has been released from its turns and convolutions may cause the cystic artery to break, tear or leak. If the cystic artery slips under the common hepatic duct (or over it) you are in trouble. Most injuries, strictures or obstructions to the common hepatic duct occur at this unfortunate moment.

10. Do not clamp or tie the cystic artery until you have convincingly identified its parent artery — usually the right hepatic.

11. Do not hesitate to expose and examine the common duct whenever

feasible. It may obviate another operation for the removal of common duct stones.

12. Do not close the common duct or tic the cystic duct stump in choledochotomy, when exploration of the common duct is completed, unless you place a catheter in the common duct via the cystic duct stump or a T-tube in the common duct. In acute cholecystitis, when exploration of the common duct is indicated drainage of the biliary tree may prevent cholangitis, acute pancreatitis or biliary dyskinesia. You might leave an unsuspected calculus in the common duct or Ampulla of Vater—even if the operative cholangiogram is normal.

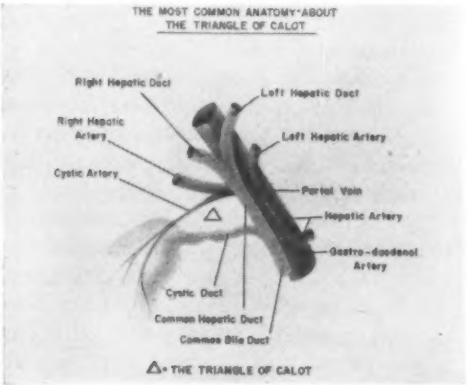

Figure 6 *

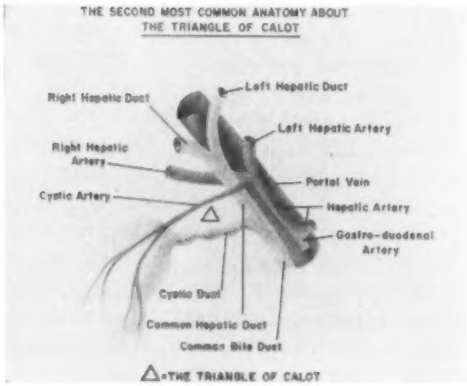

Figure 7 *

In Figures 6 and 7 are presented the schematic outlines of the two most frequently observed relations of the right hepatic and cystic arteries. Great care must be exercised often in denuding the cystic artery (with long fine pushers) so that the elbow or curve of the right hepatic artery is well visualized, in order not to include the elbow in clamp or tie. Postoperative shock, liver necrosis or atrophy have been reported when such an accident has occurred (Lahey and Pyrtek [46]).

REPORT OF A SERIES OF 97 CASES OPERATED UPON FOR ACUTE CHOLECYSTITIS

The following report records the experience of a single operator from 1939 to 1954 inclusive (16 years)—a series of 97 cases. This is an average of about 6 cases a year. Until recent years a four day (ninety-six hours) limit was rigidly adhered to, but since 1950, an occasional patient has been operated upon after ninety-six hours when all the preoperative criteria, as outlined previously, were met. Ninety-five cholecystectomies and 2 cholecystostomies were performed. Deaths were recorded in 2 cases, both cholescystostomies, and both performed under intercostal procaine anesthesia. One patient, 94 years old, blind, deaf, and in uremia (NPN 190 mg.) presented a palpable mass of two weeks' duration. A totally gangrenous, black friable gall bladder was drained. He died four days later in coma and anuria. The second patient, 58 years old, with long standing cirrhosis of the liver and ascites, acutely ill four days before admission, was operated upon the day following, after preliminary study and preparation. A distended gall bladder, easily palpable upon admission, was thought to be enlarging before surgery. Under intercostal procaine anesthesia he was operated upon through a transverse three-inch incision well below Mayo-Robson's point. A tremendously enlarged, distended, yet thick-walled acutely inflamed gall bladder, without stones, was seen. The fundus only was handled during surgery and a Pezzer catheter inserted. His wound, small as it was, disrupted com-

* The triangle of Calot was named by Jean Francois Calot in a booklet which appeared in Paris in 1891, published by G. Steinheil, and it was called "De la Cholecystectomie, (Ablation de la Vesicule Biliaire)," pages 40-42.

pletely on the seventh postoperative day with the cotton sutures cut through in all layers. Wound friability was marked. No fibrin nor coagulum was seen on secondary closure. He died on the fifth day following his second operation with a clinical picture of the so-called hepato-renal syndrome. Autopsy revealed no stones or obstruction of any type. The sphincter of Oddi was quite patent. The medical literature was investigated, searching for acute cholecystitis associated with cirrhosis of the liver. No cases were found.

THE AGE FACTOR

Sanders[69] first called attention to the fact that surgical mortality is higher in patients past fifty years of age. Both deaths in this series conform to his findings. Numerous papers since, Wallace and Allen, Glenn[34] and others, also pointed out that the younger patient has fewer complicating disorders, but the older patient usually has harbored infection in, and possibly obstruction to, the biliary tract for a long period of years, and thus has sustained varying degrees of liver damage. Compounding liver damage are cardiovascular, pulmonary, and urological handicaps that may prove fatal, during or following surgery. Table 1 outlines the age distribution and the mortality rate.

TABLE 1
AGE FACTOR AND MORTALITY RATE—97 PATIENTS
(1939 to 1954 incl.)

Decade	No. Patients	Deaths
10-20	5	0
21-30	8	0
31-40	14	0
41-50	13	0
51-60	29	1*
61-70	14	0
71-80	9	0
81-90	4	0
91-100	1	1**

* Cholecystostomy only—Age 58—advanced cirrhosis with ascites

** Cholecystostomy only—Age 94—gangrene, with uremia

It will be noted that over two-thirds of the patients were past 50 years of age with the greatest number of patients operated upon in the sixth decade. The overall mortality rate was 2.06 per cent. The fact that none of the cholecystectomized or choledochotomized patients died emphasizes that early surgery need not be too hazardous. Kirschner long ago pointed out, as have Root and Priestley[65] and others, the edema about the gall bladder and its environs often enhances easier dissection. The youngest patient was 17, the oldest 94. In the late teens and between 20 and 30 young multipara predominated. There were 69 females and 28 males in the series.

GALL BLADDER PATHOLOGY IN ACUTE CHOLECYSTITIS

The pathology of acute cholecystitis, as seen at surgery in this small series revealed a succulent, variably enlarged, diffusely congested, tense, edematous gall bladder. Since the majority of these patients were operated upon within forty-eight hours after onset the pathology, fortunately, was limited to the gall bladder, its ampulla and cystic duct. Often lines of demarcation on the neck or cystic duct could be discerned, especially in impactions in Hartmann's pouch or just proximal to the Heisterian valves. In this series there were distinctly more ampullary obstructions due to large stones than cystic duct obstructions with smaller stones. Homans' observations were similar. One ampullary stone and its wall was adherent to the portal vein! It is needless to add that the lady still has a small mural attachment of ampulla on the ventro-lateral wall of her portal vein. There were five patients in whom no stones were found or reported by the pathologists.

TABLE 2
GALL BLADDER PATHOLOGY IN ACUTE CHOLECYSTITIS
(1939 to 1954 incl.)

Acute diffuse cholecystitis	68
Acute diffuse cholecystitis with pericholecystitis, suppurative	12
Acute diffuse cholecystitis with mural abscess and infarction	5
Acute diffuse cholecystitis with perforation	5
Acute diffuse cholecystitis with empyema	2
Acute diffuse cholecystitis with solitary large infarction	1
Gangrene (complete) of gall bladder	4
No stones found	5

Two patients revealed stones within the liver substance, where perforation had

occurred in the wall, in the fossa vesicae. In one patient small stones were found within the protective inflammatory space between the gall bladder, the transverse mesocolon and duodenum. In two other perforative cases the solitary stone in Hartmann's pouch remained solidly wedged in place.

The great majority of the "easy" cases were found in the acute diffuse cholecystides, from twenty-four to seventy-two hours (acute diffuse cholecystitis). Gangrene was found in only 3 of the twenty-four to seventy-two hour groups—all in the third day of symptoms.

The concept of urging patients in the throes of acute gall stone colic to be admitted to the hospital for surgery was reborn (Kirschner, Miller, Root and Priestley and others) after repeatedly observing the early picture of acute diffuse cholecystitis. Though patients are frequently operated upon during their very first attack of gall bladder disease, Glenn stresses the fact that very few patients with acute cholecystitis fail to give a long history of symptoms relative to the biliary tract. "The patient and the gall stone fight it out to a finish" to quote Homans.[43] Surgeons might "intervene in that battle" at a time most propitious both to patient, who is acutely ill and suffering severe pain, and to surgeon, who will find a more than relatively diseased organ with superimposed acute edema and congestion.

Gangrene was found to be total in 4 patients—all past 60 years of age, and 3 contained impacted ampullary stones. Though no stones were found in 5 patients, the obstruction of the cystic duct by intense inflammation and edema was marked.

THE DURATION OF THE DISEASE AND THE MORTALITY RATE

The question of any type of surgery in the poor risk group arises in patients admitted to the hospital several days after onset. In the same group would fall those patients with known cardiovascular disease, diabetes, severe jaundice, the markedly aged and debilitated, no matter which day of the disease they enter. However,

TABLE 3
DURATION OF DISEASE AND MORTALITY RATE

Time of Operation	No. Patients	Deaths	Mortality Rate
Early (Before 96 hours)	90	0	0
Delayed (After 96 hours)	7	2	28.57%

in the delayed group there are found those in whom risk of surgical morbidity or mortality is minimal. As pointed out previously, surgery may be done at any time during the course of the disease provided the patient is well prepared for it. Before ninety-six hours, complications are not the rule, rather the exception. (Table 4). Age is an important factor. The older the patient the more associated disease and debility are found. Jaundice and common duct stone are seen in the long drawn out cases of biliary tract disease of many years standing. Cholangitis, pancreatitis, duodenitis and liver involvement are seen also in the latter decades. Especially in the delayed cases does one find more patients in the older groups. Their symptoms, as well as signs, are often minimal before serious disease is recognized. The 2 deaths reported here occurred past 50 years of age.

LOCAL COMPLICATIONS

There were few complications seen in contiguous or related organs and structures, other than common duct stone and jaundiced tissues in the patients operated upon within forty-eight hours after onset (Table 4). In the third day group gangrene of the gall bladder was found in 3 patients all past 60 years of age, but no involvement of contiguous organs other than a speckling cyanotic type of inflammatory reaction so common in the walling off process.

The pathologist often returns an acute gall bladder report with the picture of chronic cholecystitis predominating. He sometimes considers the acute elements as minimal, or even subacute. Hence, what is observed by the operator, at surgery, coupled with the clinical picture, might prove to be more vivid and actual. The edema and congestion at the cystohepatic

TABLE 4
COMPLICATIONS SEEN AT SURGERY

Common duct distention (explorations)	28 (28.87%)
Common duct stones	21 (21.65%)
Jaundice, clinical or lab. findings	19 (19.59%)
Hypertrophic lymphadenitis (cystohepatic)	11 (11.34%)
Acute pericholecystic hepatitis	5 (5.15%)
Perforation of the gall bladder (liver stones—2)	5 (5.15%)
Marked pancreatitis, acute	1 (1.03%)
Marked duodenitis, acute, adherent to G. B.	1 (1.03%)
Gall bladder adherent to portal vein	1 (1.03%)
Localized peritonitis (inflammatory mass)	Frequent
Free perforation with biliary peritonitis	None

angle of the gastrohepatic omentum is always present. The contiguous inflammatory picture produced by taxis of the neighboring organs and greater omentum cannot be seen by the pathologist. Nor does he see the mild duodenitis, pancreatitis and extrahepatic cholangitis accompanying the lesion, and so often the fine fibrinous cementing of all structures in the right upper quadrant.

In 28 cases there was sufficient evidence (jaundice, distended and dilated extrahepatic ducts, enlarged nodes along the common duct, and actually palpated stones in the common duct) to warrant choledochotomy. Common duct stones were found in 21 or 21.65 per cent of patients. Enlarged lymph nodes (other than the sentinal node) were found in 11 patients—some mimicking common duct stone in the inflammatory picture. Single or multiple stones in the common duct do not mean jaundice always, where a severe acute lymphadenitis may cause clinical jaundice. Jaundice, visual or by serum bilirubin determination (quantities over 3 mg. per 100 cc.) occurred in 19 patients.

The most frequent picture noted was the inflammatory mass surrounding the gall bladder, produced by the edema and congestion of the neighboring organs and greater omentum. It was typical of well localized peritonitis.

Since no liver biopsies were taken, the varying degrees of chronic hepatitis, cirrhosis, fatty infiltration, or even acute contiguous hepatitis only could be grossly determined. Acute contiguous hepatitis, in the partially enveloping fossa vesicae, was marked in 5 patients. This seems to be a rather meager ratio, considering the oft quoted "rich venous and lymphatic interchange" between the liver and the gall bladder wall (see section on Pathogenesis).

PREOPERATIVE AND POSTOPERATIVE NOTES

1. In addition to the criteria outlined previously, all patients are typed and matched for 2 units of blood preoperatively. Transfusion is given only if indicated.

2. Antibiotic therapy is given routinely, beginning as soon as the diagnosis is established. Therapeutic, not homeopathic nor prophylactic dosages of penicillin and streptomycin are given, and continued after surgery until the temperature is normal at least seventy-two hours.

3. Ace bandages are applied soon after the diagnosis is established—from the toes to the midportion of the calf muscles, and kept in position with longitudinal one-inch adhesive strips (Ochsner[61]). Since 1947, when, coupled with *early* ambulation, this method has been employed, no postoperative phlebothrombosis has occurred in the author's major surgery.

4. Early ambulation means many things to many surgeons. To the majority it denotes getting the patient out of bed and on his feet, walking, the day *after* or two days following surgery. Here it is defined as assisting the patient to his feet just as soon as the anesthesia effects are overcome. It is a matter of hours and not days. Instructions to the patients are given *before* surgery. Cooperation is universal and most surprising, with preliminary instruction. Marked debility and severely ill cardiac patients are the only exceptions.

5. Duodenal suction siphonage is started before surgery and continued for at least twenty-four hours post-

operatively.

6. Operative cholangiography should be used, as in elective choledochotomy if time and the patient's condition permit. It may rescue the patient from another surgical onslaught.

7. Elastic bandages and light dressings should be used upon the abdominal incision. Normal respiration requires a vast assist from the abdomen and the abdominal musculature. Cast-like adhesive bandaging leads to atelectasis, pneumonitis and poor cardiopulmonary exchange with its resultant hypoxia and cyanosis.

SUMMARY AND CONCLUSIONS

1. A short history of the development of surgery in acute cholecystitis has been sketched. Along with this development, the evolution of the ultimate procedure—cholecystectomy with drainage, is the operation of choice agreed upon by most surgeons. When the operator is confronted with an extremely critically ill patient, and he deems surgical interference imperative to save life, cholecystostomy is the procedure to be chosen, because of its simplicity and ease of accomplishment. "He who fights and runs away lives to fight another day."

2. The infectious factor, in the pathogenesis of acute cholecystitis, does not begin to explain the dramatic picture of the disease as observed in situ. It is a factor nevertheless not to be minimized, especially in the complications of perforation of the gall bladder, in gangrene and in empyema. Observations of many surgeons and pathologists lead one to believe that the involvement of the four vascular systems, entering or existing in the narrow confines about the triangle of Calot, biliary, lymphatic, venous and arterial, blocked by mural spasm and edema in the cystic duct or ampulla of the gall bladder, produces the initial and progressive pathology found. The cystic artery, acting very much like an end-artery, produces a problem in hydrostatics often impossible for the venous and lymphatic drainage to cope with. Hence, the unrelenting edema, congestion, hypoxia, diapedesis of cells and hemorrhage. Finally complete blockage of the cystic artery may occur, with resulting anoxia, gangrene and perforation. Due to the increasing intracystic pressure, the apex or ventral surface of the fundus is deprived of its blood supply first—and it is here where dissolution and perforation occur more frequently.

3. The "proper timing" of surgery in acute cholecystitis has been discussed and the evaluation of Orr (previously propounded by Wallace and Allen; Ross, Boggs and Dunphy; and DeCamp, Ochsner et al.) that "operation is indicated as soon as the patient can be prepared, provided the diagnosis is reasonably well established and there are no serious contraindications to operation". Where the critical period had been arbitrarily arrived at, between four to twelve days after onset, (because of the increased mortality ratio in that period) this lethal period exists, it is believed, because of the increasing severity of the disease rather than operative intervention.

4. Technical difficulties will always be found in biliary surgery. In early acute cholecystitis the usual cholecystectomy is made easier by the dissecting edema found about the cystohepatic angle and the triangle of Calot. Anomalies of the vessels, especially the right hepatic artery and the resultant displacement or changed position of the cystic artery, are not infrequent. The best assistant, the best anesthesiologist, the best surgical light, and the best eyesight produce a combination for the surgeon who is "at home" in surgery of the biliary tract—to reduce catastrophes so often reported, and more often bruited about in the surgical corridors. Securing, ligating, and cutting free the cystic artery first is a practice that will obviate accidents to the common hepatic duct. Cholecystectomy may be contraindicated not only in late disease but in earlier disease where a frozen area about the cystohepatic angle has resulted from previous attacks; where severe cirrhosis of the liver, pancreatitis, duodenitis etc. are encountered.

5. A report of 97 patients, operated upon by the author (1939 to 1954 inclusive) is presented. The majority (90) were operated upon within ninety-six hours. There were 2 deaths, both in patients operated upon under stress, and seriously ill. Cholecystostomy was done in both, under intercostal analgesia. One died in uremia, the other with an hepatorenal syndrome.

The ages in the series ranged from 17 to 94 years. Young mothers and multipara predominated the 17 to 30 year old group. The sixth decade represented the largest group (29). Over two-thirds of the patients were past 50 years of age. There were 69 female and 28 male patients in the series. The overall mortality rate was 2.06 per cent.

6. The elements of acute cholecystitis are often overshadowed by the predominance of chronic disease in the organ, as seen and reported by the pathologist. What is observed by the operator however is a picture of localized peritonitis, edema and congestion at the cystohepatic angle and an inflamed and enlarged gall bladder. He sees also the mild duodenitis, pancreatitis and extrahepatic cholangitis. This local pathology, plus the clinical diagnosis, with fever, leukocytosis, localized pain and/or mass, vomiting and a history of previous attacks of a similar nature comprise the surgeon's criteria—not the microscopic slide's verdict alone. This is not the fault of the pathologist, for he must "call them as he sees them". In the early stages, the first twenty-four to thirty-six hours, edema and congestion, plus chronic disease could easily be the predominant feature in any section of the gall bladder wall—especially where no infectious process is present.

Common duct explorations were carried out in 28 cases, with stones being found in 21. In 11 cases marked hypertrophic lymphadenopathy, some with jaundice, was recorded. Choledochotomy was performed when (1) a definite history of jaundice; (2) jaundice, clinical or laboratory; (3) stones palpated or seen in the common duct; (4) distended or dilated extrahepatic ducts; (5) cholangitis or (6) pancreatitis were present. Operative cholangiography should be done, if feasible.

THE CHOLECYSTIC CREED

1. Conservative therapy in acute cholecystitis is a snare and a delusion. Though it may appear immediately successful it eventually proves disastrous.

2. Successive attacks of acute cholecystitis lead to progressive inflammation.

3. Progressive inflammation leads to liver damage, pancreatic and duodenal involvement, perforations and peritonitis.

4. Patients with chronic cholecystitis are greater surgical risks when acute cholecystitis appears in their history—and vice versa.

5. Acute cholecystitis is a surgical emergency.

6. The operation of choice—cholecystectomy with drainage.

7. Jaundice, common duct stones and cholangitis require choledochotomy.

REFERENCES

1. Alexander, E. G.: Acute perforation or rupture of the gall bladder, Ann. Surg. 86:765, 1927.

2. Andrews, E.: Pathologic changes of diseased gall bladders, a new classification, Arch. Surg. 31:767, 1935.

3. Andrews, E., Schoenheimer, R., and Hrdina, L.: Arch. Surg. 25:796, 1932.

4. Babcock, R. H.: Chronic cholecystitis as a cause of myocardial incompetence, a report of thirteen cases, J.A.M.A. 52:1904, 1909.

5. Bartlett, R. W.: Bilateral intercostal nerve block for upper abdominal surgery, Surg. Gynec. & Obst. 71:194, 1940.

6. Bell, L. G., Brown, R. B., and Lenhardt, H. F.: Acute pneumocholecystitis, Ann. Surg. 138:268, 1953.

7. Berk, J. E.: The Treatment of Acute Cholecystitis: Current Therapy, Saunders, Philadelphia, 1955, P. 191.

8. Best, R. R.: The acute gall bladder, Surg. Gynec. & Obst., 73:312, 1941.

9. Bisgard, J. D., and Baker, C. P.: Pathogenesis of cholecystitis, cholelithiasis and acute pancreatitis, Ann. Surg. 112:1006, 1940.

10. Bland-Sutton, Sir. John: Brit. Med. Jour. 11:795, 1924.

11. Bockus, H. L.: Gastroenterology, Vol. III, Saunders, Philadelphia, 1946, P. 539.

12. Boyd, W.: Surgical Pathology, 6th Ed., Philadelphia, 1947, P. 320.

13. Breitwieser, E. R.: Electrocardiographic observations in chronic cholecystitis before and after surgery, Am. J. M. Sc. 213:598, 1947.

14. Brewer, G. E.: Some practical points in the anatomy of the gall bladder region, Med. News. 82:821, 1903.

15. Browne, E. Z.: Variations in origin and course of the hepatic artery and its branches; Importance from surgical viewpoint, Surgery. 8:424, 1940.

16. Bruggeman, H. O.: The treatment of acute cholecystitis, Ann. Surg. 87:423, 1928.

17. Cady, J. B., Shallenberger, P. L., and Koloski, E. L.: Cholecystocardiac disease, Guthrie Clin. Bull. 17:171, 1948.

18. Carter, R. F., Greene, C. H., and Twiss, J. R.: Diag-

nosis and Management of Diseases of the Biliary Tract; Lea and Febiger, Philadelphia, 1939, P. 378.

19. Clifford, W. J.: Acute gangrenous cholecystitis, New Eng. J. Med. 241:640, 1949.

20. Colp, R., and Doubilet, H.: Clinical significance of pancreatic reflux, Ann. Surg. 108:243, 1938.

21. Deaver, J. B.: Ann. Surg. 84:379, 1926.

22. DeCamp, P. T., Ochsner, A., Baffes, T. G., Bancroft, H., and Bendel, W.: Timing in the surgical treatment of acute cholecystitis, Ann. Surg. 135:734, 1952.

23. Denton, J.: The mode of origin of gall bladder lesions, Arch. Surg. 14:1, 1927.

24. Diffenbaugh, W. G., Sarver, F. E., and Strohl, E. L.: Gangrenous perforation of the gall bladder, Arch. Surg. 59:742, 1949.

25. Dunphy, J. E., and Ross, F. P.: Studies in acute cholecystitis—I. Surgical management and results, Surgery 26:539, 1949.

26. Eliason, E. L., and Stevens, L. W.: Acute cholecystitis, Surg. Gynec. & Obst. 78:98, 1944.

27. Enderlin, E., and Holtz, G.: Arch. f. Klin. Chir. 126:264, 1923.

28. Flint, E. R.: Abnormalities of the right hepatic, cystic and gastro-duodenal arteries, and of the bile ducts, Brit. Jour. Surg. 10:509, 1923.

29. Flint, E. R.: Gall stones associated with anomaly or injury of bile ducts, Brit. M. J. 1:995, 1924.

30. Frank, H. A., and Orr, T. G.: Early and delayed operation for acute cholecystitis, Amer. Surgeon. 19:1058, 1953.

31. Glenn, F.: Surgical treatment of acute cholecystitis, Surg. Gynec. & Obst. 73:649, 1941.

32. Glenn, F., and Moore, S. W.: Gangrene and perforation of the wall of the gall bladder, Arch. Surg. 44:677, 1942.

33. Glenn, F.: The surgical treatment of acute cholecystitis, Surgery 23:397, 1948.

34. Glenn, F.: The surgical treatment of acute cholecystitis, Surg. Gynec. & Obst. 90:643, 1950.

35. Glenn, F.: Conditions of the biliary tract requiring the consideration of early surgery, S. Clin. N. America 35:393, 1955.

36. Graham, Evarts A., et al.: Diseases of the Gall Bladder and Bile Ducts, Philadelphia, Lea and Febiger, 1928, P. 400.

37. Graham, H. F., and Waters, H. S.: Important factors in the treatment of acute cholecystitis, Ann. Surg. 99:893, 1934.

38. Hallendorf, L. C., Dockerty, M. B., and Waugh, J. M.: Gangrenous cholecystitis, a clinical and pathological study of 100 cases, S. Clin. N. America 28:979, 1948.

39. Hertzler, A. E.: Surgical Pathology of the Gastrointestinal Tract, Lippincott, Philadelphia, 1936, P. 98.

40. Heuer, G. J.: The factors leading to death in operations upon the gall bladder and bile ducts, Ann. Surg. 99:6, 1934.

41. Heuer, G. J.: Surgical aspects of acute cholecystitis, Ann. Surg. 105:758, 1937.

42. Homans, J.: Discussion of paper of Judd and Phillips—Acute cholecystic disease, Ann. Surg. 98:777, 1933.

43. Homans, J.: Textbook of Surgery, Chas. C. Thomas, Springfield, Ill. 6th Ed. 1945, P. 878.

44. Judd, E. S., and Phillips, J. R.: Perforation of the gall bladder in acute cholecystitis, Ann. Surg. 98:339, 1933.

45. Kirschner, M.: Zentralbl. f. Chir. 51:83, 1924.

46. Lahey, F. H., and Pyrtek, L. J.: Experience with the operative management of 280 strictures of the bile ducts, Surg. Gynec. & Obst. 91:25, 1950.

47. Lester, L. J.: Acute cholecystitis, with special reference to the occurrence of jaundice, Surgery, 21:675, 1947.

48. Linder, W., and Morse, L. J.: Ann. Surg. 99:357, 1929.

49. Lipshutz, B., and Kaplan, L.: Colon bacillus septicemia associated with acute cholecystitis, Surgery 10: 730, 1941.

50. Lobingier, A. S.: Necrotic edema of the gall bladder, Surg. Gynec. & Obst. 53:395, 1931.

51. Lyons, J. H., and Judd, E. S.: Collected Papers—Mayo Clinic 15:180, 1923.

52. McCallig, J. J.: Appendicular obstruction; its clinical and pathological aspects, Surg. Gynec. & Obst. 73:539, 1941.

53. McKibbon, J. P., and McDonald, J. R.: Surgery 17:319, 1945.

54. McLaughlin, C., Jr., and Raines, M.: Obstruction of the alimentary tract from gall stones, Am. J. Surg. 81:424, 1951.

55. Magner, W., and Hutcheson, J. M.: Cholecystitis, bacteriological and experimental study, Canad. M.A.J. 27:469, 1932.

56. Mentzer, S. H.: Acute cholecystitis, its surgical treatment, Calif. & West. Med. 32:224, 1930.

57. Mentzer, S. H.: The acute gall bladder manifesting few signs or symptoms, Surg. Gynec. & Obst. 55:709, 1932.

58. Miller, R. H.: Ann. Surg. 90:644, 1930.

59. Moosman, D. A., and Coller, F. A.: Prevention of traumatic injury to the bile ducts: A study of the structures of the cystohepatic angle encountered in cholecystectomy and supraduodenal choledochostomy, Amer. J. Surg. 82:132, 1951.

60. de Massy, G.: Quoted by Revilont; Gaz. d. hap. 1878, P. 566.

61. Ochsner, A.: Venous thrombosis, J.A.M.A. 132:837, 1946.

62. Pines, B., and Rabinovitch, J.: Perforation of the gall bladder in acute cholecystitis, Ann. Surg. 140:170, 1954.

63. Potter, A. H.: Biliary disease in young subjects, Surg. Gynec. & Obst. 66:604, 1938.

64. Rehfuss, M. E., and Nelson, G. M.: Am. J. Digest. Dis. & Nutrition 1:759, 1935.

65. Root, G. T., and Priestley, J. T.: Acute cholecystitis, certain pathologic and surgical aspects, Am. J. Surg. N.S. 61:38, 1943.

66. Rosenow, E. C.: Collected Papers of Mayo Clinic 8:22, 1916.

67. Ross, F. P., and Dunphy, J. E.: Studies in acute cholecystitis II—Cholecystostomy, indications and technique, New England J. Med. 242:359, 1950.

68. Ross, F. P., Boggs, J. D., and Dunphy, J. E.: Studies in acute cholecystitis III—The pathological process in relation to the clinical management of the disease, and the fallacy of the "critical period", Surg. Gynec. & Obst. 91:271, 1950.

69. Sanders, R. L.: Perforation of the gall bladder, Surgery 1:949, 1937.

70. Smith, B. C.: Acute cholecystitis, surgical treatment of 332 cases, S. Clin. N. America 25:285, 1945.

71. Smith, M. K.: The treatment of acute cholecystitis, Ann. Surg. 98:766, 1933.

72. Stone, H. B., and Owings, J. C.: The acute gall bladder as a surgical emergency, Ann. Surg. 98:760, 1933.

73. Strohl, E. L., and Diffenbaugh, W. G.: Acute cholecystitis, S. Clin. N. America 32:63, 1952.

74. Tendler, M. J.: Acute appendicitis with rupture, a report of 689 cases, Memphis, M. J. 1951.

75. Thorek, M.: Surgical Errors and Safeguards, J. B. Lippincott, Philadelphia, 1932, P. 378.

76. Touroff, A. S. W.: Acute cholecystitis, a study of 75 proven cases with subsiding clinical manifestations at time of operation, Ann. Surg. 99:900, 1943.

77. Van Zwalenburg, C.: Acute obstructive appendicitis, its mechanisms, J.A.M.A. 42:820, 1904.

78. Wallace, R. H., and Allen, A. W.: Acute cholecystitis, Arch. Surg. 42:762, 1941.

79. Walters, M. B., and Master, A. M.: The relationship between biliary tract and coronary artery disease, Surg. Gynec. & Obst. 94:152, 1952.

80. Walton, A. J.: A Textbook of the Surgical Dyspepsias, London, E. Arnold & Co., 1923.

81. Weiss, M. M., and Hamilton, J. E.: The effect of gall bladder disease on the electrocardiogram, Surgery 6:893, 1939.

82. Whipple, A. O.: Bull. N.Y. Acad. Med. 7:211, 1931.
83. Wolferth, C. C.: Relationship between biliary tract disease and heart disease, J. Mt. Sinai Hosp. N.Y. 8:1121, 1942.
84. Wolfson, W. L., and Rothenberg, R. E.: Acute non-calculous cholecystitis, J.A.M.A. 106:1978, 1936.
85. Zinninger, M. M.: Ann. Surg. 96:406, 1932.

DISCUSSION

Dr. Robert Kapsinow, (Lafayette): As I read over the copy of the paper by the essayist, I was amazed at the prodigious amount of literature he must have reviewed and abstracted. From personal experience, I can assure you it was no easy task and for that alone, he should be congratulated. It is nevertheless a curious fact that there are as many of our great surgical leaders arrayed on the side of conservative as there are on the side of radical surgery. Therefore, the problem resolves itself, not into accepting the opinions of one camp or the other, but into a high degree of individualization, not only of each patient but of the medical philosophy of the surgeon.

With each new advance in medical science, there comes a change in some phase of therapy in medicine or surgery, because of the advance. The magnitude of our thinking and our technics become ever more spectacular and more radical. So it has been with the subject of the acutely inflamed gall bladder. Electrolytes, hydration, transfusion and antibiotics have won new converts. The foreboding implications of such terms as gangrene, perforation and biliary peritonitis create a real panic.

As stated by the essayist and others, most acutely inflamed gall bladders arise because of an impaction of a calculus either in the cystic duct or the ampulla of the gall bladder. Bile can no longer get into the gall bladder and with the arrest of drainage, the circulation of the gall bladder becomes impaired and edema results. Several authors have attested to the small amount of bile spillage that results from perforation of the gall bladder and it is not evidence that the patient would have died, had he not been operated upon.

The constantly repetitious comparisons between the acutely inflamed appendix and the gall bladder are erroneous. The function, physiology and bacterial flora are different. Why not compare it with the acutely inflamed salpinx.

It occurs to me that the thinking should be along the line of whether jaundice is present or not. In the presence of jaundice, the chance of subsidence of the acute process is less problematic and inasmuch as the common duct will invariably have to be explored, then the sooner the abdomen is opened, the better. When jaundice is not present, then it becomes a matter of whether the condition is worsening or improving.

The concept of delayed surgery does not mean that the patient is left untreated. Indeed the extreme opposite is the truth. The very electrolytes, transfusions, fluids, and antibiotics used to prepare the patient for what must always be considered as emergency surgery, are exploited to the fullest, but with a plan conceived to tide the patient over the crisis to a time when ideal dissection and less hazardous exploration of the common duct may be undertaken.

Doubilet, in a different paper than the one quoted by the essayist, reports a series of 116 cases of acute cholecystitis which were treated by withholding surgery until the optimum time for operation. After excluding 19 cases which were treated for acute pancreatitis as the etiological cause, 55 cases received interval cholecystectomy. Of the entire series, only one patient died. Ten or 18 per cent of those undergoing surgery showed evidence of previous perforation or gangrene.

For the acutely ill rapidly deteriorating gall bladder patient, surgery must always be considered and all concerned should accept it as a calculated risk. It takes a great deal of courage to do a cholecystostomy immediately and quickly as a life saving procedure "and come back to fight another day" than to start doing a cholecystectomy and find it to be impossible and settle for a cholecystostomy.

I would like to make this query. Obviously not all the acutely inflamed gall bladders that have come under the essayist's care, were operated upon. I wonder what happened to those who refused immediate surgery?

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SIGNIFICANCE OF OVERWEIGHT IN ETIOLOGY, PROGNOSIS, AND TREATMENT OF DIABETES MELLITUS, WITH REPORT OF CASES *

A. A. HEROLD, SR., M. D.
SHREVEPORT

Ever since Newburgh and Marsh of Ann Arbor, 1920, in the preinsulin days, advocated a high fat diet in diabetes, there has been much discussion as to calories, fats in food, and other problems in therapy of this affliction.

Allen in this country and Guelpa in Italy had, in the meantime, advocated undernutrition and advised starting this antiketotic and weight reduction technique by eliminating, first, fats and then proteins and carbohydrates, substituting low-nutritive liquids, notably broth, and whiskey. This idea was rational and had many followers, augmented constantly, until the advent of insulin in 1922.

* Presented at the Seventy-fifth Annual Meeting of the Louisiana State Medical Society, New Orleans, May 4, 1955.

I feel that insulin usage, generally speaking, is not a cure for diabetes, but symptomatic relief and I am convinced, further, that its use, over a considerable period of time, especially in children, may be actually harmful, by resting the islets of Langerhans too much (possibly resulting in atrophy) and thereby making the patient more and more dependent upon the exogenous product, with the probability that a mild case might be converted into a severe one. How, then, is it possible to *cure* the disease? The answer is by dietary supervision; in the slender patients, careful study of the requirement with moderate increase in carbohydrates and, for a while, at least, giving proper insulin to promote metabolism and, very important, not to overlook the part being played by the liver, which, if damaged, causes overproduction of sugar in system through a faulty glycogenic function.

But with the obese diabetic, the problem is different, as we shall illustrate by a few cases from our records; weight reduction is the key and many patients, properly regulated for gradual loss of avoirdupois, may be taken off of insulin and caused to live comfortably provided the weight is held down.

This brings us to a consideration of the etiological factors. There can be no doubt about the part played by heredity. Whenever there is a history of the disease in the family of one parent, there is, according to authorities, a 50-50 chance that one descendent in 4 will develop it; if both parents' families have had it, one-half of the offspring will be pretty sure of getting it. Then, there is the probability of some focal infection resulting in pancreatitis and, ultimately, diabetes, especially in cases of gall bladder disease and pyelitis.

The third—and very important factor—is obesity and this fault is usually the result of overindulgence in food. Proper reduction of the overweight diabetic is the key.

Medical literature abounds in references to this subject and I shall quote a few, after which I will give synopsis of cases in our experience at North Louisiana Clinic in Shreveport.

In quoting authorities, I might first refer to many excellent articles in various issues of *Diabetes*, notably Vol. 2, No. 6, Nov. -Dec. 1953 number; Drazin writes on *Glucose Tolerance in Hypertension and Obesity*, concluding with the observation that both conditions decrease glucose tolerance, especially when they are concomitant, but more marked in the obese. These observations were on nondiabetics, only.

Short in *Medical Arts and Sciences*, (Vol. 6, No. I) on *Obesity*, notes that glucose tolerance is related to overweights, finding in 10 to 19 per cent overweight, an impairment of 18 per cent; whereas in 50 to 59 per cent overweight, it amounts to 44 per cent.

W. H. Olmstead in *Michigan State Journal* (Vol. 52, No. 10) in an article entitled *Obesity, with Key to Prevention of Diabetes*, states that undernutrition, as advocated by Allen should be kept in mind when advancing age develops obesity; to quote: "Obesity precedes the onset of diabetes in 85 per cent of individuals past 40 years."

T. P. Sharkey in *Ohio State Journal*, Nov. 1953, under subject of *Diabetes Mellitus and Obesity*, states: 1st, Heredity determines susceptibility to diabetes; 2nd, statistics show obesity to be the most important predisposing factor; 3rd, more prevalent in urban areas, due to less physical exertion; 4th, insulin contraindicated, as a rule, in most obese diabetics, except for complications. (Of Course!)

Newburgh and Conn (*J.A.M.A.* Vol. 112, No. 1) call attention to the various causes of hyperglycemia and glycosuria, but stress the obesity factor. Their conclusions: Gastineau and Ryneason (*Annals of Int. Med.* Vol 27, No. 6) stress the seriousness of the dangers of handicaps of obesity and urge that correction in both diabetics and nondiabetics should depend upon limitation of calories and not on drugs. On the other hand, Roberts (*Annals of Int. Med.*, Vol. 34, No. 6) advocates the use of dexamyl as a valuable ad-

junct in reducing overweight diabetics, so that they may reduce the glycosuria and glycemia, as many cases have done and thereby eliminate insulin.

Quoting from Duncan on *Diseases of Metabolism* (p. 717 and p. 721):

"Those most widely recognized are (a) heredity, (b) endocrine disturbances affecting the pituitary gland and adrenal cortex, and to a less extent the thyroid gland, (c) obesity, and (d) a group of miscellaneous factors such as race, sex, infection and destructive lesions of the pancreas." . . .

"Note should be made of the recent papers by Haist, Campbell and Best, who have shown that fasting or a diet high in fats leads to a reduction of the insulin content of the pancreas while a diet high in carbohydrate leads to an increased content."

CASE REPORTS

W. L. G. A. aged 63, first consulted me in December 1947, with a typical history of uncontrolled diabetes; weight was 280½ lbs., excessive appetite, had eliminated sweets but paid no attention to total calories. Complications have been psoriasis and arthritis; also prostatic disease. Under supervision of diet (which has not been very satisfactory), all troubles have cleared and he, now, with blood sugar readings near normal and weight 257 lbs. or lower, feels very well and is aglycosuric.

E. H. A. aged 42 when he first consulted me (Oct. 1952) with chief complaint of diabetes known since 1935, and now with furunculosis for past year. His highest weight had been 271 lbs., at this time 209 lbs.; symptoms, polyuria, polyphagia and polydipsia; blood sugar 216 with 3 plus glycosuria; blood pressure 142/90. He stated that he had been on a diet, but not like I gave him. Pyorrhea noted and ordered corrected.

Nov. 1952, weight 204, urine 1 plus.

Dec. 1952, weight 196, urine 1 plus and clear.

Apr. 1953, weight 189, urine clear and faint trace; B.P. 139.

July 1953, weight 190, urine all clear; B.S. 113, B.P. 120/82.

Boils gone and feeling fine. Later reports the same.

F. A. aged 63 in 1950. First seen by me with chief complaint of diabetes (as far as he knows) for four weeks previously; usual symptoms and loss of some weight. Has had bad teeth (and pyorrhea) which is being attended to. Weight 186½ lbs., formerly a little over 200 lbs. Polyuria, polydipsia. Has been put on 17 units P.Z.I. elsewhere. Shortly thereafter, I left the city and he was cared for by Dr. Herold, Jr., who followed him along dietary lines, gradually withdrawing insulin. When last seen, he was taking no insulin, weight down to 166, blood sugar 127, feeling well and this has continued to date, to the best of our knowledge.

Mrs. J. M. B., age 57, first had symptoms of diabetes in 1951, at which time she had showed increased blood sugar at times. She visited two very prominent clinics in this country and reported to me in March 1951, with weight of 189 lbs., blood pressure 170/94 and taking 20 units insulin daily and receiving, in return, many hypoglycemic reactions. With reduction of diet, gradual elimination of insulin, her weight came down, last blood sugar record being 160 postprandial, weight 162½ lbs., blood pressure 142/88 and feeling well.

These are but four of our cases picked at random; we have many more similar ones in our files. You will note that none of these are now on insulin and, while I strongly advocate insulin, when indicated, I think that patients can, in such instances, do very well without it.

And now, as a brief summary and a few personal impressions, I would like to conclude, as follows:—

1. As metabolism of fats, without presence of carbohydrates, produces ketone bodies, we should be careful, in dietary menus, not to eliminate carbohydrates entirely.

2. Never neglect the liver's need for carbohydrates, for, with exhaustion of glycogen, this organ may throw out toxic substances, which develop into ketone bodies, as shown by experiments of Mirsky and associates. Also the work of Soskin emphasizes the importance of the liver (over-production) in diabetic etiology.

3. Insulin not only metabolizes sugar, but it also enables the liver to store it as glycogen.

4. Slender diabetics, practically all — like growing children — require insulin with proper control of calories, but with liberal carbohydrates.

5. All juvenile diabetics require insulin but care should be taken not to get reactions and I advise against useless glucose tolerance tests.

Finally, obesity is the main thing to

avoid in those with diabetic tendency or diabetic family history and the principal weapon to combat it with is caloric restriction.

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EVALUATION OF ONE YEAR'S EXPERIENCES WITH CULDOCENTESIS AT CHARITY HOSPITAL OF LOUISIANA AT NEW ORLEANS *‡

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Among the diagnostic aids in the armamentarium of the gynecologist, none has been more valuable on the Tulane service at Charity Hospital than culdocentesis. Employed in hundreds of cases, the procedure has proved itself to be quick, innocuous, and ultimately accurate, when interpreted properly. The term "culdocentesis" was coined nine years ago by Beacham [1] to describe the aspiration of fluid from the cul-de-sac or pouch of Douglas in the female. At that time, culdocentesis may have been viewed by many to be used only on hospitalized patients as a teaching procedure. Today, it is employed by most gynecologists on the Tulane staff and other practitioners on patients in the office, at home, in outpatient departments and on wards when the diagnosis of a case is doubtful. Culdocentesis is especially valuable as a diagnostic aid. It should be remembered that culdocentesis is not only responsible for many accurate diagnoses leading to emergency surgery, but conversely, many patients are spared an unnecessary laparotomy.

METHOD OF CULDOCENTESIS

With the patient in the lithotomy position, the vagina and cervix are painted with a mild, bacteriostatic solution, such as aqueous zephiran. A Graves speculum is inserted. The posterior lip of the cervix is then grasped with a tenaculum or sponge-holder (we find the latter preferable, since it usually will not cause bleeding). The cervix is mobilized anteriorly and caudad, so as to expose the posterior fornix. An 18 gauge, long spinal needle is then placed lightly against the vaginal mucosa, in the midline of the posterior fornix and about 1 cm. lower than the insertion of the uterosacral ligaments. The needle is then pushed firmly and quickly about 2 cms. into the cul-de-sac. (The patient experiences moderate, momentary, transient pain when the needle perforates the peritoneum, but it is so mild that anesthesia or analgesia is rarely required. We have tried local anesthetics such as pontocaine and cocaine earlier in the series and have not found even this procedure necessary. We have, on occasion, given 100 mgms. demerol I. M. or I. V. for analgesia to a hyperkinetic patient, and it has helped some and complicated others by causing vomiting or stupor. By and large, our culdocenteses are done without anesthetics or analgesics.) A glass adapter, attached to an aspirating syringe by means of a small length of rubber tubing, is then inserted into the needle and suction is made with the syringe. In the absence of the glass adapter with rubber tubing, the syringe may be attached directly to the spinal needle and the contents of the cul-de-sac aspirated. The needle is not pointed laterally, is held steadily in the midline and is not permitted to "wander". It is withdrawn in the same fashion as it is introduced. One tap frequently produces enough fluid to establish a diagnosis—but if no fluid is obtained, a "dry tap" is recorded and the procedure is repeated once or twice.

EVALUATION OF CUL-DE-SAC FLUID

There are four categories of fluid that may be aspirated:

1. Blood
2. Pus

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* Presented at the Seventy-fifth Annual Meeting of the Louisiana State Medical Society, New Orleans, May 3, 1955.

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‡ Aided by the Ex-Residents and Fellows' Research Fund.

3. Peritoneal fluid
4. Encysted fluid (e.g., dermoid, endometrioma, follicular cysts)

The above may occasionally be found in combination, especially if the needle point is inserted too deeply or too far laterally.

1. *Blood*:

A. When blood is aspirated from the cul-de-sac, it should be observed for at least six minutes. If it does not clot, it is pathognomonic of hemoperitoneum and the patient should be regarded as a surgical emergency and operated upon as soon as practicable. The majority of patients in this category will be found to have ectopic pregnancies. A small number of patients will have bleeding from ovarian cysts. These cysts are usually ruptured follicular or corpora luteal cysts. Nonclotting blood may contain particles of old blood clots, which is especially diagnostic of hemoperitoneum.

B. If the blood clots, it may be assumed to have come from an artery, vein, or the uterus and the entire procedure should be repeated with a new culdocentesis set.

C. Pink-tinged peritoneal fluid (not blood, is also indicative of a "traumatic tap".

D. "Watery" blood aspirated is indicative of a mixture of blood and peritoneal fluid; here, experience in interpreting culdocenteses is important, as well as consideration of the clinical picture.

2. *Pus*:

Aspiration of free pus indicates peritonitis or ruptured viscus. (e.g., ruptured tubo-ovarian abscess, ruptured appendix, ruptured gall bladder, or acute pancreatitis).

A. Aspiration of encysted pus indicates a pelvic abscess. If the mass bulges into the posterior fornix, it is our policy to establish the diagnosis by culdocentesis, aspirating approximately 20 cc. of pus and then instilling 10 cc. of varidase through the same spinal needle into the abscess, in order to decrease the viscosity of the abscess contents. This is followed in twenty-four hours by colpotomy. (If the mass is encysted and out of the pelvis, we do not attempt aspiration). The pus obtained is smeared and examined directly, then sent to the laboratory for culture and sensitivity studies.

B. A diagnosis of ruptured pelvic abscess classifies a patient as a surgical emergency. Immediate surgical management of such patients in the past three years has reduced the mortality rate from ruptured pelvic abscess on our service from 60 to 10 per cent.[8] Credit for the rapidity of diagnosis must be given to culdocentesis.

3. *Peritoneal Fluid*:

When this type of fluid is obtained clear on aspiration, a ruptured viscus is not present. Of course, if clinical reason points to an unruptured tubal pregnancy, the patient may be hospitalized for twenty-four to forty-eight hours for direct observation, *blood typing and cross matching and repeated culdocentesis.* Or she may be sent home and instructed to return quickly if abdominal pain, bleeding or syncope ensues. *Blood typing and crossmatching is still mandatory.*

4. *Encysted Fluid*:

A. Dermoid fluid, which is yellow and congeals on cooling, may be obtained accidentally if a dermoid is not suspected. Abdominal removal of the dermoid solves this problem.

B. Chocolate fluid aspirated from an endometrioma establishes a diagnosis of endometriosis.

C. An ovarian cyst, suspected of being a persistent follicular or corpus luteal cyst, and revealed by its clear, straw-like color, may be emptied by culdocentesis and save the patient an abdominal operation.

D. If a cystic mass is strongly suspected of being malignant, direct aspiration is contraindicated. However, it may be possible to aspirate peritoneal fluid from the cul-de-sac and this fluid may be centrifuged in the laboratory, the sediment imbedded in wax, sectioned and examined cytologically for metastatic, malignant cells. This method, plus biopsy of a random inguinal node on the affected side,

originated by Collins, is employed on the Tulane service in attempting to diagnose carcinoma of the ovary.

COMPLICATIONS OF CULDOCENTESIS

It is our belief that the procedure of culdocentesis is essentially simple, so much so that senior medical students in Gynecology at Tulane are required to perform at least one before being graduated. It is regarded as just as fundamental as teaching a student to biopsy or cauterize a cervix or teaching him how to perform an adequate pelvic examination. Interpretation of the aspirated fluid requires some experience—but the presence of nonclotting blood to the novice or professional means hemoperitoneum and emergency surgery is indicated.

Culdocentesis may be considered by some to be a dangerous procedure but we are of the opinion that it is innocuous. Bowel has, on several occasions, been entered through the cul-de-sac with the spinal needle. The latter has been withdrawn without "wandering" the point and a new set of instruments used to repeat the procedure. Of all the culdocenteses performed on our service, there are no recorded or verbal instances of peritonitis, fistulae, or pelvic abscesses developing as a result of this inadvertent entry into the bowel.

REVIEW OF MORTALITY OF ECTOPIC PREGNANCY

The mortality due to ectopic pregnancy at Charity Hospital, uncorrected, was 11.4 per cent from 1919 to 1936 (51 deaths). From 1937 to 1950, the mortality was 2.6 per cent (18 deaths). From January 1, 1951, to July 1, 1953, the mortality was 0.38 per cent (1 death in 255 cases of eccyesis. This patient died on arrival before admission.) Of these cases, 133 were on the Tulane service. Including the latest 45 cases in this report, there were 178 patients with proven ectopic pregnancies from January 1, 1951, to July 1, 1954, who were treated surgically and successfully on the Tulane service with *no* fatalities. Credit must be given to the improved residency system, better anesthesia and blood banks but the major factor contributing to the lowered mortality in this entity has been culdocentesis.[2-7]

REPORT ON THE ONE YEAR STUDY OF CULDOCENTESIS

Beginning with July 1, 1953, a detailed record of each culdocentesis performed in the admitting rooms and clinics was kept by the residents on the Tulane Unit, Charity Hospital. Special forms for this purpose were devised and mimeographed and included the following data. Patient's name, age, race, hospital number or home address, type of fluid obtained, number of taps per patient, disposition, diagnosis, and treatment. Each resident signed his name and date on these forms, which were collected daily and totalled for each month. Monthly totals were added in computing the yearly totals. Although the residents were responsible for the statistics, the culdocenteses were performed by various personnel:

1. Staff
2. Residents and Fellows in all levels of training.
3. Internes [Supervised by (1) or (2)]
4. Medical students [Supervised by (1) or (2)]

In the year surveyed (July 1, 1953, to July 1, 1954), 13,338 patients (9,970 colored and 3,368 white) were seen on the Tulane service at Charity Hospital in the clinics and admitting rooms (Table 1). Of these, 717 patients (613 colored and 104 white) had culdocenteses performed. (Table 2). That is to say, for every 18.6 patients seen, 1 had a culdocentesis. The total number of single "taps" done was 1,088, which means that each patient had an average of 1.5 cul-de-sac aspirations. It should be re-emphasized that these were

TABLE 1
CULDOCENTESIS
TULANE UNIT—CHARITY HOSPITAL
JULY 1, 1953 - JULY 1, 1954

Total Patients Seen		
	W. 215	
Admit Room	C. 739	954
	W. 3153	
Clinics	C. 9231	12,384
Total		13,338

TABLE 2
CULDOCENTESIS
TULANE UNIT—CHARITY HOSPITAL
JULY 1, 1953 - JULY 1, 1954

Culdocentesis Performed		
Admit Room	W. 40 C. 228	268 Patients (28%)
Clinics	W. 64 C. 385	449 Patients (3.6%)
Total		717 Patients (1,088 single "taps")
(13,338 Pts.)	(5.4% had culdocentesis)	

not indiscriminately selected patients, but they were patients who presented signs or symptoms of possible ruptured viscae which, according to our criteria, prompted the cul-de-sac punctures.

Of the 717 patients who had culdocentesis performed, 59 yielded nonclotting blood. In 30 patients pus was aspirated, peritoneal fluid was obtained in 527 patients and 101 demonstrated no fluid (Table 3). Of the 59 patients (8.2 per cent) who had nonclotting blood on culdocentesis, 3 were not operated (2 therapeutic aspirations of vaginal cuff hematomas and 1 endometriosis). The remaining 56 were operated (Table 4). Of these (Table 5), 45 had proven ectopic pregnancies; 3 had a diagnosis of bleeding corpus hemorrhagicum; 3 had bleeding corpora luteal cysts and 1 had a hemorrhgic tubo-ovarian mass.

TABLE 3
CULDOCENTESIS
TULANE UNIT—CHARITY HOSPITAL
JULY 1, 1953 - JULY 1, 1954

Results	
1. Blood	59
2. Pus	30
3. Peritoneal fluid	527
4. Dry	101

TABLE 4
CULDOCENTESIS
EVALUATION OF ACCURACY

Patients with Unclotted Blood	59
Minus Patients Not Operated (Includes 1 Endometriosis & 2 Therapeutic Aspirations of Vaginal Cuff Hematomas)	3
Patients Operated with "Positive" Cul de Sac Punctures	56

TABLE 5
CULDOCENTESIS

	Correct Diag.
Pts. with Ectopic Preg.	45
Bleeding Corp. Hemorrhag.	3
Bleeding Corp. Luteum Cysts	3
Hemorrhage into Both Adnexae	1
Hemorrhagic T-O Mass	1
Total	53
ACCURACY: $\frac{53}{56} = 94.6\%$	

There were 3 patients who demonstrated no bleeding foci at laparotomy (Table 6), so that 53 patients out of 56 were operated upon with the correct diagnosis. This represents an accuracy of 94.64 per cent for the entire year. There were no fatalities in any of the above cases.

Of the 30 patients (4.2 per cent) who had pus on culdocentesis (Table 7), 4 had

TABLE 6
CULDOCENTESIS

	Incorrect Diag.
Threatened Abortions	2
Salpingo-Oophoritis	1
Total	3

TABLE 7
CULDOCENTESIS
ANALYSIS OF 30 PTS. WHO YIELDED PUS

Ruptured T-O Abscesses	4
True Pelvic Abscesses	15
Pelvic Peritonitis	3
Salpingo-Oophoritis	6
Old Pyosalpinx, Asymptomatic	1
Dermoid Cyst	1
Total	30

(Above pts. treated medically or surgically with no fatalities.)

ruptured tubo-ovarian abscesses treated by immediate surgery; 15 had unruptured cul-de-sac or tubo-ovarian abscesses. The cul-de-sac abscesses were treated by colpotomy and the tubo-ovarian abscesses (which did not "point") were treated medically until they were "cooled down", then they were treated surgically after an appropriate interval had elapsed. Culdocentesis greatly aided in the proper selection of an antibiotic, since the pus ob-

tained was smeared directly, then sent to the laboratory for culture and sensitivity studies. Three patients had pelvic peritonitis; 6 had acute salpingo-oophoritis; 1 had an old asymptomatic pyosalpinx and 1 had a dermoid cyst of the ovary. All these patients were treated medically or surgically and there were no fatalities.

Clear peritoneal fluid was revealed on culdocentesis in 527 patients (73.5%) which was considered as normal (Table 3).

No fluid was obtainable on aspiration of the cul-de-sac in 101 patients (14.0%) and these were listed as dry.

We would like to point out that many of the above patients had been seen by other physicians, both inside and outside the hospital, before admission to our service. The diagnosis for each one of these patients was made or confirmed by the use of culdocentesis. We feel certain that had culdocentesis been employed by these other physicians, the correct diagnosis would probably have been made. All physicians can perform culdocentesis — at home, in the admitting room, and in the office. Most physicians do not possess a culdescope—but they do have a needle and syringe. Patients do not have to be subjected to general anesthesia and exploratory laparotomy or colpotomy if physicians will use culdocentesis to help them establish or confirm their diagnoses.

CONCLUSIONS

1. Culdocentesis is especially valuable in confirming a diagnosis of hemoperitoneum or establishing one where it was previously unsuspected.

2. Culdocentesis may be done on outpatients as well as inpatients. Basic equipment includes an 18 gauge long, spinal needle and a hypodermic syringe.

3. Culdocentesis helps establish a diagnosis more rapidly; hence, morbidity is diminished, mortality is lowered, and number of patient days in the hospital are decreased.

4. Culdocentesis is an invaluable aid in treating peritonitis and pelvic abscesses since the aspirated pus can be sent to the laboratory for culture and sensitivity studies, resulting in the selection of the proper antibiotic.

5. Culdocentesis is innocuous, shown by the absence of recorded or verbal complications in the 717 patients who had 1088 single "taps" in the surveyed year.

6. All physicians can perform culdocentesis at home, in the admitting room and in the office. Most physicians do not possess a culdoscope—but they do have a needle and syringe.

(This statistical study was consummated only through the splendid cooperation of the entire Tulane Ob.-Gyn. Resident Corps and we wish to acknowledge publicly the immense help they contributed to the project.)

REFERENCES

1. Beacham, W. D., Collins, C. G., Thomas, E. P., and Beacham, D. W.: Ectopic pregnancy, J. A. M. A. 136:365 (Feb. 7) 1948.

2. Graffagnino, P.: Ectopic pregnancy, Am. J. Obst. & Gynec. 4:148 (Aug.) 1922.

3. Tyrone, C. H., Romano, S. A., and Collins, C. G.: Tubal gestation; Statistical study based on 309 cases, Am. J. Obst. & Gynec. 30:112 (July) 1935.

4. Collins, C. G., Beacham, W. D., and Beacham, D. W.: Ectopic pregnancy. Mortality and morbidity factors, Tr. Am. A. Obst., Gynec. & Abd. Surg. 59:141, 1948.

5. Beacham, D. W., and Beacham, W. D.: Culdocentesis, New Orleans M. & S. J., 103:283 (Jan.) 1951.

6. Collins, C. G., Beacham, W. D., Beacham, D. W., Griffon, J. P., and Nix, F. G.: Ectopic pregnancy, Clin. Med. Soc. Bull. (Chicago Medical Society) 54:357 (Nov.) 1951.

7. Johnson, C. G., and Post, L. I.: Ectopic pregnancy, Surg. Gynec. & Obst. 98:481 (April) 1954.

8. Nix, F. G., Nelson, E. W., and George, J. H.: Bull. of Tulane Univ. Med. Fac., 13:179 (August) 1954.

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LOUISIANA PHYSICIANS: PIONEERS IN THE AMERICAN PUBLIC HEALTH MOVEMENT*

BEN FREEDMAN, M. D., M. P. H. †
NEW ORLEANS

In 1934, this Society commemorated at its annual gathering, the centennial of the establishment of the Medical College of Louisiana, later to become the Tulane School of Medicine. In 1944, it celebrated the centenary of the founding of the New

* Presented at the Seventy-fifth Annual Meeting of the Louisiana State Medical Society, New Orleans, May 3, 1955.

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Orleans Medical and Surgical Journal. Both of these events were notable milestones in the development of medical knowledge in Louisiana. At this meeting we signalize the hundredth year since the founding of the Louisiana State Board of Health. We can justly honor this occasion also for several reasons. Firstly, this is the oldest State Board of Health in the United States. Secondly, its founding was the handiwork of the physicians of Louisiana, struggling against powerful forces to create an instrument for the control of epidemic disease based on the application of the most enlightened medical knowledge of the time. Thirdly, the physicians responsible for its creation, and subsequently, for its progressive development were among the most eminent practitioners and teachers in the history of medicine in Louisiana, many of whom were nationally famous, playing brilliant roles as medical and public health pioneers of our country. It is fitting, therefore, at this time to recall some of the important social forces and events, and the roles of some of these eminent physicians whose genius and energy led to the establishment of the State Board of Health and helped to shape the history of the public health movement in America.

The history of public health in Louisiana can be understood only in the context of the history of public health in the United States and in colonial America, and this in turn, only as a continuation of the public health history of the old world.

THE PRELUDE

Every period in human history has been characterized by the pre-eminence of a particular pattern in the incidence of diseases. This was so because of the very nature of social change. From one period to the next, social and environmental changes had inevitably taken place. Each period, therefore, was a unique medium for sustaining the occurrence and spread of certain diseases in a certain pattern.

The disease patterns which had predominated in the prehistoric epochs have not as yet been fathomed. It does, however, appear that the first diseases to terrorize civilized man were the diarrheas and dysenteries. At the dawn of civilization, man already lived in large enough communities whereby the contamination of the soil and water by excreta could have given rise to epidemics of enteric diseases. It is quite probable that such epidemics began to occur among the barbarians of the upper neolithic period even before civilization, when organized village life first came into existence. The rationale of the above is based on the fact that no human physiological activity is more conducive to the ubiquitous spread of disease than that of defecation. Man has had thousands upon thousands of years to ponder this problem. That he did engage his faculties to consider this problem is evident from the fact that excreta control practices were already well developed at the beginning of recorded history. At that time, privy and drainage construction of good quality were found in excavated villages of the Middle East. Such ingenious inventions must have taken hundreds and even thousands of years of experience to develop in those days when social change and the coming into being of new ideas took so long. These inventions came about not from knowledge of the causes of enteric fever, but from experience and empirical consideration that such practices served as safeguards against illness. It can be safely presumed, therefore, that the basic lessons in hygiene for these ancients came not from the pedagogy of their magicians and medicine men, but from their terrible experiences with diarrhea and dysentery.

By the time that the biblical period came upon the scene of history, social conditions had changed enough to alter the pattern of the disease experience of man. His previous experience, however, became incorporated in the Mosaic Code of hygiene practices in the first five books of the Old Testament. This code plainly indicates a long standing concern with practices aimed to eliminate the hazard of diarrheas and dysenteries. It appears that

during the biblical period, various contagious and infectious diseases plagued community life, such as leprosy, ophthalmia, infantile epidemics, pestilential fevers, enteric infections, venereal diseases, elephantiasis, skin diseases and many others, but the disease which spread the greatest terror among the people was no longer the enteric infections, but leprosy. This disease became the dictator and taskmaster of the biblical and dark age periods. This was the tyrannical pedagogue who taught with mortal chastisement, the techniques of isolation, and decontamination—measures which also served well in the control of many other communicable diseases.

As the clouds of the Middle Ages began to disperse, a new and restless epoch emerged, animated by science and pressured into commercial and exploratory enterprise. Thus, a new pattern of social development was initiated, bringing forth a new complex of diseases to try man's endurance and to test his ingenuity. Among these diseases one stood out as the sign of the times, to wit, the plague. "A more frightful teacher than the plague, which swept over humanity with special fury in the middle of the fourteenth century, it is difficult to imagine," is a deftly chosen quotation lifted by Winslow [1] from Diepgen's essays. Concerning this apt description of the situation, Winslow [2] further comments, "As suggested in the quotation . . ., the Black Death was the great teacher in this field (i.e., in the epidemiology of infectious diseases.) A preliminary course was, however, offered by leprosy and, at the end of the fifteenth century, a post-graduate course by syphilis."

COMMUNITY EXPERIENCE AS TEACHER

The student of the history of medicine and public health may become intrigued by the finding that the origins of the basic practices used by civil authorities to control epidemic disease have seldom resulted from discoveries made by the great figures in medical history, but rather were learned by communities from the experience of tragic visitations of various epidemic diseases. The student must, therefore, be struck by the following facts:

1. That the fundamentals by which communities protect the lives of their people were developed very early in the experience of mankind.

2. That these *basic* practices appear to have been developed by community efforts rather than by individual ingenuity.

3. That few physician's names, or the names of other persons have been found associated with the discovery of any of these *basic* practices.

4. That mostly in the refinement of techniques do we find associated the names of great physicians or other professional men, and this more usually in modern times.

Not only were the basic environmental and personal hygienic techniques such as disposal of excreta, control of water supplies, draining of swamps, control of insects and rodents, protection of food, avoidance of overcrowding, decontamination of fomites, isolation and quarantine, and the cleansing of hands and body discovered anonymously through community experience, and mostly in the deep past, but also such fundamental techniques as sterilization and vaccination. Only with regard to the refinement of these techniques, and their extrication from the perplexity of empiricism and their elevation into a scientific sphere, are the names of great men associated.

THE AMERICAN EXPERIENCE

The forebears of the American colonists, therefore, received their most poignant lessons in the preservation of health from such teachers as leprosy, syphilis, and the plague. The latter disease almost destroyed the commerce that made its spread possible. But the great teacher [3] of the colonists themselves and the descendants for three hundred years was another plague, the yellow fever—an epidemic terror which struck awe in the hearts of Americans until the genius of the Carlos Finlay-Walter Reed team finally ferreted out the last secret from this celebrated pedagogue.

Why was yellow fever such an effective teacher? Undoubtedly because, like plague, it struck like lightning with devastating effects, leaving a population stunned with consternation and expectancy. And also because the nature of its spread could not be wholly described on the basis of the usually accepted dynamics of contagiousness and infectiousness. It required the most versatile of mental gymnastics, of philosophic acumen, of observational acuity, and of statistical analytics to try to solve the mystery of its mode of occurrence and spread. Every phase of the physical and social environment had to be taken into the scope of deliberation in developing a rationale of its epidemiology. Besides all this, the medical profession was completely helpless in treating this plague, and are not today exceptionally versatile in this matter.

Yellow fever was first accurately described by Jean Baptiste Dutertre [4] in Guadaloupe, French West Indies, in 1635. It, however, took some years before knowledge of this disease entity became widely known. According to Dr. Henry R. Carter [5], preponderant evidence indicates that this disease was imported to America from Africa during the early slave trade period. By the middle of the 17th century the disease appears to have become well established in the West Indies, and had moved into Central and South America. A series of yellow fever epidemics in Yucatan, Barbados, and Cuba in 1648-49 had repercussions farther north. In Massachusetts, the general court, meeting in Boston, established a precautionary quarantine, in March 1648, for all vessels arriving from the West Indies because of "ye plague or like infectious disease" [6] which was not lifted until the following year in May 1649. The outbreak in the West Indies having been taken for a plague-like disease, the usual sanitary measure employed against plague by the Europeans, namely, quarantine, was invoked.

There is no evidence that any yellow fever occurred on the North American continent before the last ten years of the 17th century. What was probably the first yellow fever outbreak on the North American continent occurred in 1693, when a British fleet coming from Barbados anchored in Boston harbor. Concerning this, Cotton Mather wrote: "In the month of July a most pestilential Feaver, was brought among us by the Fleet coming in to our Harbour from the West Indies. It was a Distemper, which in less than a week's time usually carried off my Neighbours, with very direful Symptoms, of turning Yellow, vomiting and bleeding every way and so Dying." [7] In 1699, an epidemic in Philadelphia destroyed one-sixth of the inhabitants. In the same year a severe epidemic appeared in Charleston and also again in 1703, 1728, 1732, 1739, and 1740. Philadelphia had epidemics in 1741, 1747, 1748, 1762. The most panic-ridden epidemic prior to the 19th century was in Philadelphia in 1793, where La Roche estimated that about 11,000 cases occurred with over 4,000 deaths from a population of about 50,000 persons. At that time Philadelphia was the capitol of Pennsylvania and of the United States. The panic was so great that the central processes of administration of the Governments of the United States, of Pennsylvania, and of Philadelphia were in complete dissolution for the duration of the epidemic.

American medicine in the 17th, 18th, and most of the 19th century, was based on the classical Hippocratic-Galenic teachings with various degrees of modern influence as represented mainly by the teachings of Sydenham, Boerhaave, Cullen, Monro, Lettsom and Brown. For preventing yellow fever, this wisdom was very unproductive, although potentially fruitful with its concept of "epidemic constitution." For treating this disease, it was either neutral or harmful, but practically always void of virtue.[8] Acute, easily communicable diseases with rapid mortalities have always been the bane of the therapeutic art. This is almost as true today as it was then, and particularly so for yellow fever. No wonder this disease

became the great teacher in American medicine and in the health administration of civil government. Community catastrophe that could not be controlled by treatment had to be conquered by prevention. To this end, the scientifically trained American physicians of the 18th and 19th centuries devoted the greatest of their mental efforts. One needs only scan the American medical literature of these two centuries, and follow the story of the physicians' participation in the development of public health administration in North America to realize how cogently true this was.

THE LOUISIANA EXPERIENCE

The early experience of North America in health and disease was, to a great degree, the experience of Louisiana. New Orleans was founded in 1718. Yellow fever was believed to have existed there practically every year up to the date of American domination in 1803,[9, 10] but with epidemic intensity in 1765 and 1766. During the colonial period, there were evidently but few physicians in New Orleans. Most of these had immigrated from Europe, and appear to have been of French and Latin extraction. They were as highly trained as European schools could render, and had the exquisite culture of gentlemen of the day. As far as the available records show, their participation in civil government in the role of medical sanitarians is not easily detected. Only after annexation to the United States when American trained physicians migrated into Louisiana, did a trend of participation in community health problems become noticeable among physicians. This latter observation pertaining to the early lack of the physicians participation in community affairs may be more apparent than real. Because of the natural terrain and meteorological conditions, environmental hygiene could not but have been a most important consideration of medical men of Louisiana. During the 19th century, New Orleans experienced more than 60 outbreaks of yellow fever.

Because of the persisting epidemics, Governor Claiborne, in 1804, urged the Legislative Council to consider a scheme of city planning suggested by the President of the United States, Thomas Jefferson. Several years later, Claiborne recommended the adoption of quarantine and general health laws. Apprehensive of a prevailing yellow fever epidemic situation, the city council of New Orleans on June 4, 1816,[11] called a meeting to obtain the advice of the Société Medicale. The Public Health Committee of the society gave the city council a long list of recommendations, most of which pertained to the sanitation of the city. Among the recommendations was the creation of a Board of Health. Because the opinion among the Société Medicale was strongly toward the theory of the importation of yellow fever, adoption of quarantine laws was also recommended. In the winter of 1816-17, an act creating a Board of Health and Quarantine laws was passed by the State Legislature. This law was quite comprehensive, and the regulations were enforced by the police. However, the State appointed body incurred the displeasure and antagonism of the city authorities. Consequently, an Act was passed on March 6, 1819, abolishing the Board of Health, health officer, and quarantine laws, but the governor was invested with the right to establish quarantine by proclamation. In 1821, a severe yellow fever epidemic caused similar laws to be adopted again, and the Board of Health was reinstated. The laws, however, were poorly framed and feebly administered.

The value of an effective quarantine was clearly demonstrated in 1822. Quarantine existed at the river entrance to the city, but none was in vogue from the lake side. Yellow fever was raging at Pensacola. Two boats carrying yellow fever cases from that port, not being able to enter the city through the river, entered through the Lake side by way of Bayou St. John and introduced the epidemic into New Orleans. A protest meeting was held, in 1823, by citizens who believed that the quarantine regulations were injuring the city's commercial develop-

ment. In 1825, the pressure of public opinion aroused by commercial interests resulted in a repeal of the quarantine laws. The City Council, however, was given the right to appoint a health officer and to establish quarantine inside of New Orleans, and the governor was given quarantine powers. Between 1820 and 1830, yellow fever appeared in New Orleans every year, about half of these years in epidemic prevalence. During the next decade, the epidemics of 1832, 1833 and 1839 were disastrous. With the discontinuance of quarantine in 1825, New Orleans apparently had no organized health supervision until 1841. In 1835, however, a strong medical society was in operation, and two physicians were detailed from this body as advisors to the Mayor.

PHYSICIANS FORCE THE CREATION OF A STATE BOARD OF HEALTH

It was at the beginning of the 1830-40 decade that one of our country's most illustrious physician-sanitarians, and certainly the first eminent one in Louisiana, began his rise. From his first published paper in 1832, which is considered Louisiana's first medical classic [12], entitled "The Application of Physiological Medicine to Diseases in Louisiana," Dr. Edward Hall Barton's acuity of observation and perspective, scholarly passion for knowledge, and fortitude in the service of his community destined him to become one of the most distinguished medical scientist-philosophers and men of action of his day. In 1832, he also made a most portentous observation [13] about yellow fever which appeared to be the reason why he later kept insisting on the drainage of the swamps around New Orleans to rid the city of mosquitos. In citing the remark of the 17th century Dutch anatomist and epidemiologist, Diemerbroeck, concerning the uncommon abundance of insects preceding the occurrence of pestilence, Barton observed that "flies and mosquitoes particularly were unusually numerous preceding the epidemic," and that the mosquitoes continued throughout the summer. He was one of the world's pioneers in the development of medical statistics as an instrument of medical science. This work he began around the same time as that of the great French clinician, Pierre-Charles-Alexandre Louis, who is credited with having been the founder of medical statistics, as distinguished from vital statistics.

In the list of Barton's published contributions are to be found wisdom and vision which even today are inspirations to scholars. He was one of the driving forces which shaped the pioneering history of Louisiana to protect itself against epidemic onslaughts. He was the third Dean of the Tulane Medical School, between 1836 and 1840, and was the chief engineer in laying the foundation of the Louisiana State Board of Health.

The decade between 1840 and 1850 was featured by terrible ravages of yellow fever, decimating the population. But still thousands came to New Orleans, lured by the high wages and the stories of possibilities of making fabulous fortunes in a short time. At the insistence of the physicians of the city, a Board of Health and quarantine were established by the General Council in June 1841. After several years the board lost interest, despite recurring epidemics, and was dissolved in 1843. The physicians, however, continued to urge the city to create a Board for community protection, so that in 1844, the General Council invited the Medico-Chirurgical Society to take charge of the sanitary interest of New Orleans. This proposition was accepted, and a committee of nine members was appointed with full powers to act as a Board of Health. The years 1847, 1848, 1849, were among the most disastrous epidemic years in Louisiana history.

The lack of interest and reluctance to act on the part of the city fathers was traditional in New Orleans whose reputation was described by DeBow in 1846: "New Orleans, disguise the fact as we may, has had the reputation abroad of being a great charnel house." Filth and disease stalked hand in hand with death.

Such eminent physicians as Drs. E. H.

Barton, E. D. Fenner, A. F. Axson, and J. C. Simonds will remain alive in Louisiana medical annals for their tireless labors and fearless revelations of local health hazards. They counselled the city officials and endeavored to open their eyes to the grave conditions existing in this thickly populated city. They created the plans to better the conditions—plans that needed only implementation. The dauntless Dr. Barton appeared before the city council in 1850, and predicted dire calamity unless sanitary conditions were improved. His words fell on deaf ears. In January 1851, Mayor Crossman officially proclaimed the city to be perfectly healthful in spite of the fact that the death rate per 1,000 population for 1849, 1850, and 1851 was 78, 84, and 62 respectively as compared to a present day rate of 10.

In 1853, Dr. Barton's prediction unfortunately came to pass. Yellow fever was brought into the city in May. In June it picked up momentum. In July panic broke out. The merchants vigorously denied the "rumors" of epidemic and the newspapers, in collaboration, refrained from making mention of it, although during the week of July 9th, over 400 persons died and Charity Hospital became a busy depot for sending corpses to the grave. People began to flee the city by the thousands. Among them were many members of the city council which adjourned soon after July 22, 1853, belatedly recognizing the epidemic situation and hurriedly passing a resolution authorizing the formation of a Board of Health. The Board was organized on July 26, and the council adjourned on July 28. The fleeing members of the city council left Mayor Crossman to face a tragic and appalling situation. The council did not convene again until the end of the epidemic late in November. The temporary Board of Health was created with Dr. A. Hester, editor at that time of the New Orleans Medical and Surgical Journal, as physician of the port. It was, however, too late. About 30,000 cases of yellow fever occurred in New Orleans during this epidemic with over 7,500 deaths. Many more cases and deaths occurred over the entire state.

The persistent efforts of Dr. Barton, Dr. Simonds, and their fellow physicians began to bear fruit. The people of New Orleans began to learn their lesson in sanitation. The public urged the Board of Health to appoint a sanitary committee to investigate the cause of the epidemic and to make recommendations for future prevention.[14] Late in 1853, the Board of Health appointed such a commission consisting of Honorable A. D. Crossman, Mayor of the City of New Orleans, Drs. E. H. Barton (Chairman), A. F. Axson, J. C. Simonds, J. L. Riddell, and S. D. McNeil. Despite the 1853 catastrophe and the impeachment of the Street Commissioner for dereliction of duty in keeping the streets clean, the city council, in 1854, returned to its traditional apathy toward health. The disgraceful indifference occasioned in the summer of 1854 during the Council's consideration of a much needed Health Ordinance was as culpable as its final action before adjournment of ignominiously laying the Ordinance on the table.

In the face of such gross negligence as was the historically traditional mood of the New Orleans City Council, it was no wonder that Governor Hebert[15] passionately appealed to the State Legislature in 1854 for the creation of machinery for protecting the entire state against epidemic incursions. If New Orleans, the gateway to the state for the importation of disease, were morally paralyzed against acting for the well-being of its citizens and those of the state, then the state itself must perforce go into action.

The State Legislature depended completely on the physicians of the state to give direction to the legislators in the framing of a state law for the protection of the health of the people. To this end, the Senate in 1854 ordered 250 copies of Dr. Fenner's *History of the 1853 Yellow Fever Epidemic* to be printed. The 1854 Legislature, not being fully prepared to pass its health bill, laid it on the table

indefinitely. By the time of the convening of the 1855 State Legislature, the report of Dr. Barton's investigating commission was completed, and was already widely read and discussed with much passionate exchange of views between supporters and antagonists. By joint resolution of the House and Senate, 140 copies of Dr. Barton's report were ordered printed. The testimony of all physicians was pleaded for by the Joint Public Health Committee of the State Legislature in order to guide the creation of adequate and effective legislation. The majority of the physicians held the theory that yellow fever, like other dread disease epidemics, was imported, and therefore, the technique of quarantine was essential for control. Although the Barton report recommended a comprehensive sanitary program for control of epidemic disease, and the Fenner study recommended the same, the State Legislature enacted a law, signed by the Governor on March 13, 1855, entitled "An Act to Establish Quarantine for the Protection of the State." It must, however, be understood from the plea of the Governor, the intent of the House and Senate, the Majority and Minority Reports of the Joint Legislative Committee on Public Health, and the wording of the Act itself, that general sanitation authority was intended for the Board of Health, although quarantine was its emphasized activity. On June 13, 1855, the first permanent State Board of Health in the United States was selected with the eminent and colorful Dr. Samuel Choppin as chairman. Dr. Barton and two other prominent physicians also served on this board. From this date until the reorganization of 1898, New Orleans paid the penalty of being denied the authority to organize its own Board of Health and was dependent on the State Board of Health for advice in matters of public health.

THE PHYSICIANS WHO SERVED THE PUBLIC HEALTH MOVEMENT

In a century of commendable operations, and at times of brilliant achievements, many of the most famous Louisiana physicians served as presidents of the Board or as member participants. Such medical names as Samuel Choppin, A. Forster Axson, Gustavus A. Nott, Charles Delery, Charles B. White, Joseph Jones, Joseph Holt, Samuel R. Olliphant, Clement P. Wilkinson, Edmond Souchon, Oscar Dowling, and John H. Musser had graced its leadership. Among the galaxy of its members and co-workers before the turn of the century were Drs. Warren Stone, William M. Carpenter, Bennett Dowler, N. B. Benedict, Josiah C. Nott, John D. Foster, Abner Hester, H. D. Baldwin, Stanford E. Chaillé, Samuel M. Bemiss, Felix Formento, Andrew W. Smythe, George K. Pratt, Rudolph Matas, Frederick Loeber, Alfred W. Perry, Daniel C. Holliday, Stephen S. Herrick, G. Farrar Patton, Frederick W. Parham, S. C. Russell, Lucien F. Salomon, J. Charles Faget, William H. Watkins, Paul E. Archinard, Charles Chassaignac, Isadore Dyer, Ernest Lewis, and hundreds of others. It can be safely asserted that practically every reputed physician in Louisiana during the period of twenty-five years before and twenty-five years after the establishment of the Board participated in molding the forces which created it and guided its subsequent development. A complete listing of the names of these men would include most of the pioneer medical educators in New Orleans, the organizers and officers of the medical and scientific associations, the editors of the medical journals, the chief surgeons of the Charity Hospital, and men who otherwise made New Orleans famous as a medical center.

Our nationally famous state health officer, Dr. C. B. White, was one of the founders of the American Public Health Association in 1872. He served as second vice-president in 1873, as member of the Executive Committee in 1877-78-79, and was elected to presidency of that Association in 1880. Dr. Samuel Choppin was elected second vice-president in 1878 and first vice-president in 1879. In 1880, Dr. D. C. Holliday was elected to the executive committee, and during these public health

pioneering days many more of our socially oriented physicians played important roles on various committees of the Association which carried on missionary work in the science of community health. Many important papers in public health were delivered by Louisiana physicians at the annual meetings of the American Public Health Association. Dr. Felix Formento, many years a member of the Louisiana State Board of Health, chairman of its laboratory committee which was responsible for the creation of the first state board of health diagnostic bacteriological laboratory in the United States in 1894, and president of the American Public Health Association in 1892, presided as one of the presidents *d'honneur* at the International Hygiene Congress in Geneva in 1882. Dr. Stanford E. Chaillé and Dr. S. M. Bemiss were members of the National Board of Health which was created by Congress in 1878, Dr. Chaillé as its supervising inspector. Both served on the Havana Yellow Fever Commission appointed in 1879, of which Dr. Chaillé was president and of which Dr. Rudolph Matas served as secretary.

Is there any wonder therefore, considering this remarkable activity on the part of our physicians, that during the first two decades of the existence of the American Public Health Association an exceptionally large proportion of its members were physicians from Louisiana. For example, in 1884, more than 10 per cent of the entire American Public Health Association membership was composed of Louisiana physicians. Those men were highly conscious of what it took to control or to eliminate epidemic disease. The articles with which they filled the medical journals were cogent testimonials of their breadth of vision, their scientific and philosophic attainments in the field of knowledge, and the enlightened cultural background with which they approached their professional activities. This is our medical heritage of public health in Louisiana.

The State Board of Health, therefore, was created by the wisdom and fortitude of the most eminent of our medical forebears to serve a basic community need which could otherwise not be attained. This essential instrument of public service has been and will continue to be as effective as the physicians of the State will it to be.

REFERENCES

1. Winslow, C.-E. A.: The Conquest of Epidemic Disease, Princeton University Press, 1944, p. 88.

2. Ibid. p. 89.

3. Winslow, C.-E. A.: The History of American Epidemiology, C. V. Mosby Co., 1952, Chapter I, The Colonial Era and the First Years of the Republic (1607-1799).

4. Garrison, F. H.: History of Medicine, 4th ed. 1929, W. B. Saunders Co., p. 825.

5. Williams, R. C.: The United States Public Health Service, 1798-1950, p. 119, Whittet and Shepperson, 1951.

6. Duffy, John: Epidemics in Colonial America, p. 140, Louisiana University Press, 1953.

7. Ibid. p. 141.

8. Powell, J. H.: Bring Out Your Dead, University of Pennsylvania Press, 1949.

9. Report of the Health and Sanitary Survey of the City of New Orleans, 1918-19, p. 4.

10. This statement is refuted by the evidence presented by Dr. A. E. Fossier, in his article, **History of Yellow Fever in New Orleans**, The Louisiana Historical Quarterly, 34:205, 1951. The claim is made that the first epidemic occurred in 1796.

11. Baudier, Roger: Sanitation in New Orleans, Southern Plumber, Vols. 9 and 10.

12. Postelle, W. D.: Louisiana: Its records of Medical Progress, New Orleans M. & S. J. 96:530, 1944.

13. Fossier, A. E.: History of Yellow Fever in New Orleans, Louisiana Historical Quarterly, 34:213 (July) 1951.

14. Postelle, W. D.: Edward Hall Barton, Sanitarian, Ann. Med. Hist., 4:370-381, 1942.

15. Louisiana State Legislative Journals of the House of Representative and Senate, 1854.

DISCUSSION

Dr. Guy Jones (Lockport): I wish to compliment Dr. Freedman on his splendid presentation of the history of Public Health developments, and particularly, on the history of the development of the Louisiana State Board of Health, of which I happen to be a member at this time. We are rather proud of the fact that the Louisiana State Board of Health is 100 years old this year, and is the oldest State Board of Health in our nation.

As Dr. Freedman has pointed out in giving his references to the illustrious gentlemen who founded and served on the State Board of Health, these men were principally practicing physicians who recognized the need for a Board which would have the authority by law to require sanitation and to institute preventive measures in controlling and preventing disease epidemics.

One hundred years ago, diseases of all kinds were very prevalent throughout our State. Yellow fever occurred in New Orleans as late as 1905. I am happy to note at this time that many

of the diseases which we used to see, even when I began practicing medicine a few years ago, have become very rare. We have not had a case of yellow fever since 1905. Typhoid fever and malaria, which used to be so common, are very rare today. There can hardly be any doubt but that the efforts of public health in the field of environmental sanitation, mosquito control, and improvement of our water, food, and milk supplies, have played a primary role in the eradication of these diseases.

Due to the generalized practice of immunization against smallpox, diphtheria, and other communicable diseases, these diseases have become quite rare. Public health practices in the field of environmental sanitation and mass immunization, working hand in hand with the practicing physicians, have accomplished a great deal over the years in improving the health of our people.

Tuberculosis and other diseases are on the wane due to the cooperative efforts of public health people in finding new cases of tuberculosis and bringing them to the physicians for treatment.

I could refer to many more situations in which, by team work, we practicing physicians and public health people have been able to improve the health of our community.

A few years ago, the physician was seldom called except when the patient suffered pains, fever, injury, or other distressing symptoms demanding relief. By pointing out that many diseases began early before presenting readily observed symptoms, and recommending that each person consult a physician regularly, there is no doubt but that many lives are saved by seeking the advice of a physician before an incipient disease becomes far advanced and incurable.

In closing, I would like to emphasize the thought that public health is not in competition with the practice of medicine, but is a member of our team, working hand in hand with us toward the goal of a healthier and happier population.

"Public Health is a purchasable commodity, available in large or small quantities, in a variety of packages, and its value increases after purchase. It is a difficult article to place on display or the sales counter. Public Health is sometimes reluctantly bought, but once secured, is not given up. Its best salesmen are major disasters and disease epidemics".

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DIAGNOSIS OF DIABETES MELLITUS

During the coming month the attention of medical societies, physicians, and the public—to some extent—will be drawn to the problems of diabetes, primarily, diagnosis. The Diabetes Detection Drive sponsored by the American Diabetes Association is held each year in November. It is primarily an effort on the part of those physicians who constitute the American Diabetes Association to stimulate our physicians to find, diagnose, and treat as many as possible of the now one million unknown diabetics in this country. This type of activity in the public interest is in accordance with the highest traditions of medicine and well deserves our support.

In the typical—and if typical, usually advanced—case of diabetes the diagnosis is not difficult. In the majority of cases of manifest diabetes needing treatment and undetected the diagnosis is not nearly so obvious. Moss [1] has spoken of diabetes as the great imitator in modern medical practice. In an analysis of the essential facts of 140 patients with diabetes mellitus, only 12 had classical symptoms; 11 had pruritis vulvae and 9 each complained of muscle joint pains and indigestion; 7 each were concerned with routine physical examination and fatigue. The remaining 85 were concerned with 44 other conditions, none suggestively diagnostic of diabetes. Among the vast majority, therefore, of the 140 patients the diabetic condition was masked under symptoms of another syndrome, to which, no doubt, it was contributory. The importance of this is seen when one reflects upon the trend in practice today.

An increasing number of patients go direct to the specialist in whose field they consider that their complaint should be treated. The physician limiting his practice to one organ or to one system looks for organic disease. If he finds it, or if he does not, the condition is handled on its merits, and unless diabetes is specially looked for, it is easy for him not to see in the symptomatology an insidious and complicated disease which may permeate every niche of the body before the symptoms become severe enough to be readily recognized. In diabetes, as in many other conditions, the diagnosis is only found if it is specifically looked for.

The diabetic who consults the ophthalmologist may manifest his disease in a variety of ways. Aside from the vascular changes which are manifest on fundus examination, the patient may complain of blurred vision produced by acute myopia, or of oculomotor or abducens paralysis.

Involvement of the autonomic nervous system may have produced a typical Argyll Robertson pupil in patients who have never had syphilis. In a similar manner in the field of every other specialist there are syndromes which may appear to be organic behind which the precipitating factor is diabetes.

It is of particular importance to mention at this point the syndrome of acute abdomen, simulating such surgical diseases as acute appendicitis, in which actually diabetes is the cause of the symptoms. Moss mentions especially the syndrome of "postoperative diabetes," described as a condition in which the stress of surgery has precipitated clinical diabetes in patients predisposed to the disease. He recommends that all patients should have a urinalysis two days after a major operation, and cautions that sometimes the failure to diagnose diabetes is due to the assumption that glycosuria is the result of a preceding intravenous infusion of glucose.

The physician, therefore, regardless of what field in which he may practice, may render a great service to his patient by routinely suspecting the disease and undertaking appropriate steps to prove or disprove its existence.

Cumulative clinical experience has established certain standards by which the results of tests for glycosuria and hyperglycemia may be judged. Only the moderately advanced and advanced diabetics show glycosuria when fasting. For this reason, it is best that a urinalysis for sugar be taken two hours after a meal containing at least 100 grams of carbohydrate.

The American Diabetes Association considers that the following findings indicate the presence of diabetes with a high degree of probability: the presence of any amount of glucose in the urine accompanied by blood sugar values as follows:

A. Venous blood sugar:
   1. Folin-Wu method—over 130 fasting, over 200 after eating.
   2. Somogyi method, or other methods for true glucose value—over 110 fasting, over 150 after eating.

B. Capillary blood sugar:
   1. Folin-Malmros method—over 140 fasting, over 240 after eating.
   2. True glucose value methods—over 120 fasting, over 200 after eating.

In absence of glycosuria a high renal threshold may exist. Such occurs when the glomerular filtration rate of sugar is impaired more than its tubular resorption. In such a condition diagnosis would have to be made on blood sugar findings. It should be cautioned, however, that the diagnosis of diabetes should not be established upon the basis of only one high blood sugar determination. When results are inconclusive recourse should be had to glucose tolerance determinations concerning which there is considerable variance in technique and in interpretation. Most authorities classify the curve as normal if the blood sugar returns to its initial normal level within two hours.

Diabetes is a disease of increasing incidence. This is because methods are more accurate and because of the increased longevity of the population as a whole. Joslin has estimated that there are 2,500,000 persons living in the United States today who either have or will develop diabetes. Other analysts have predicted that if mortality rates continue to follow the same trend as at present, by 1980, deaths from diabetes will be exceeded only by those from heart disease.

In summary, it may be stated that diabetes is a great imitator; that it is increasing; and that its detection requires a specific effort on the part of the physician.

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[1] Moss, James M.: Diagnosis of diabetes mellitus, J. Student A.M.A. 4:19 (February) 1955.

[2] Moss, James M.: Diabetes mellitus, the great imitator of modern medical practice, Exhibit, Meeting of the A.M.A., June 6-10, 1955, Atlantic City.

[3] Joslin, Elliott P. and others: Exhibit of the Joslin Clinic, Meeting of the A.M.A., June 6-10, 1955, Atlantic City.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

ATTENTION ALL MEMBERS

Pursuant to the instructions of the House of Delegates and the Executive Committee, the results of the efforts of the Subcommittee appointed by Dr. Max M. Green, our President, to assist and cooperate with Dr. C. Grenes Cole, your Secretary, in the employment of a full time public relations individual or firm as assistant to the Secretary were reported to a called meeting of the Executive Committee on August 20, 1955.

This Subcommittee was unable to employ a satisfactory full time individual or public relations firm who would answer our needs at this time. However, this Subcommittee recommended to the Executive Committee, as a substitute, that we employ both a public relations firm and an experienced individual in legislative work to represent us for the ensuing year. This recommendation was unanimously adopted by the Executive Committee and in accordance with its instructions, the public relations firm of Perret and Kalman of New Orleans and Mr. Percy J. Landry, Jr., of Baton Rouge, as a field representative, were employed.

We sincerely believe a wise decision has been made in this matter and we respectfully request that all our members cooperate wholeheartedly with us in any programs developed for the future good of our Society, its members and the people of our State.

C. GRENES COLE, M. D.
Secretary-Treasurer

IMPORTANT INFORMATION ON ACCREDITATION OF HOSPITALS

The following are the most recent decisions of the Joint Commission on Accreditation of Hospitals, and should be of interest to staff members of all State and private hospitals.

Medical Staff Meetings are Important!

From the start of the American College of Surgeons Hospital Standardization Program in 1920, the holding of regular medical staff meetings has been one of the standards on which approval of a hospital has been based. In accepting the Standards for Hospital Accreditation established by the College, the Joint Commission on Accreditation of Hospitals endorsed this principle as sound and essential for good patient care.

The sole purpose of hospital medical staff meetings is to improve the quality of patient care by means of a critical review and evaluation of the work of the professional staff. This analysis must be done by the medical staff which should meet at frequent and regular intervals to accomplish this purpose.

Recognizing that differences exist in hospitals because of size, location, and medical staff organization, hospitals have a choice of **one** of the three following methods of fulfilling the medical staff meeting requirement:

1. **Monthly Meetings of the Entire Active Medical Staff.**
   This method is particularly suited in the hospital of less than seventy-five beds which is not departmentalized. It is also well suited in the hospital where the medical staff is small and is organized to function as a committee of the whole.
2. **Monthly Departmental Meetings and Quarterly Meetings of the Entire Active Staff.**
   This method may be adopted when a hospital is well organized and departmentalized, and when it has been found to be more effective than Choice 1 for adequate and efficient review of the work of the staff. More frequent departmental meetings will depend upon the size of the hospital and the number of patients treated. Many hospitals have found it advisable to hold weekly departmental meetings.
3. **Monthly or More Frequent Review of the Clinical Work by the Medical Records and Tissue Committees,** (or by an Audit Committee which combines the functions of these two committees), **Plus Monthly Meetings of the Executive Committee of the Medical Staff and Quarterly Meetings of the Entire Active Medical Staff.**
   If conscientiously carried out, this method is very effective in evaluating the quality of patient care.

Experience has shown that one of these three patterns of medical staff meetings will fit any hospital seeking accreditation, whether it be small or large, rural or urban, affiliated with a medical school or not.

There has been some confusion and misunderstanding among the medical profession and hospitals concerning the following aspects of hospital medical staff meetings:

1. **Frequency and Regularity of Meetings.** The regular monthly meetings as described in Choices 1, 2, and 3 with not less than twelve in each calendar year, are necessary for adequate and effective evaluation of

MEDICAL STAFF MEETINGS REQUIRED FOR ACCREDITATION

CHOICE	Meeting	When Held	Required Attendance*	Purpose and Program	Conditions
1.	Medical Staff	Monthly and not less than 12 meetings in each calendar year.	75 per cent of active staff	Review and critical evaluation of the clinical work in the hospital. Report of Committees.	Suitable in smaller hospitals.
2.	Staff Departments	Monthly or more frequently	75 per cent of active members of department	Review and critical evaluation of work of departments.	May be substituted for (1) monthly staff meeting when hospital is well organized and **all** medical work is covered in departmental meetings and CPC'S.
	Medical Staff	Quarterly	75 per cent of active staff	Report of medical work for preceding quarter presented by representatives of departments or by executive committee.	
3.	Medical Records and Tissue Committees or Audit Committee	Monthly or more frequently	75 per cent of members of committees	Review of all medical work in hospital, as stated in assignment of committee functions	May be substituted for (1) monthly staff or departmental meetings when hospital is well organized and **all** medical work is covered in committee conferences, and reports presented to staff.
	Executive Committee	Monthly	75 per cent of members of Committee	Appropriate study and action on reports of medical records and tissue committees or of Audit Committee.	
	Medical Staff	Quarterly	75 per cent of active staff	Report of medical work for preceding quarter presented by executive committee to medical staff	

* Members may be excused by the executive committee for exceptional conditions such as sickness, absence from the community or medical emergencies.

patient care. This means that such meetings should be held during the summer vacation months, for patients are treated in the hospital during these periods and their care should be evaluated.

2. **Multiplicity of Meetings.** Numerous complaints have been voiced by the medical profession concerning the hardship of forced attendance at medical staff meetings of several hospitals to which they have appointments. This occurs because many physicians accept an impracticable multiplicity of active staff memberships. There is a growing feeling among the practicing profession that a physician should limit his active staff appointments to no more than two hospitals in order that he may best serve his patients and conserve his time and energy. If additional hospital facilities are desired by the physician, he may accept an associate, consulting, or courtesy staff appointment. Administrations of hospitals should cooperate with the medical profession by making beds readily available to physicians with other than active staff appointments.

3. **Combined Hospital Medical Staff Meetings.** In certain areas where the medical staffs of two or more hospitals are identical, there may be a desire to combine the monthly staff meetings of the several hospitals into one time-saving meeting. This is not acceptable, for it defeats the prime objective of a medical staff meeting, the evaluation of the care given patients in a particular hospital.

4. **Attendance at Meetings.** Adequate and effective participation in the evaluation of patient care requires regular, conscientious attendance at meetings of the active medical staff, departments or committees. It is expected that members of the active medical staffs shall attend at least seventy-five per cent of these official meetings, unless excused by the Executive Committee of the medical staff for such exceptional conditions as sickness, absence from the community or because of medical emergencies.

5. The Standard of the Commission on the attendance of staff meetings pertains to the active medical staff members. It is the

decision of the local hospital staff whether the attendance of associate staff members is required.

6. Departmental meetings and clinicopathologic conferences are not required by the Commission, although highly desirable and recommended. These are requirements of the Council on Medical Education and Hospitals of the American Medical Association for training programs.

Remember, the requirements for medical staff meetings of the Commission are minimum for quality patient care. Many hospital staffs exceed these requirements and for this they should be commended.

TO THE MEMBERS OF THE LOUISIANA STATE MEDICAL SOCIETY:

It is through this medium, your Journal, that I invite you to set aside October 28th and 29th to attend the annual meeting of the Louisiana Health Council which will be held in New Orleans at the Jung Hotel. As you know, this organization in this state is similar to the National Health Council being sponsored by the A.M.A. and locally by the Louisiana State Medical Society.

Our program this year is built around the life cycle of man. The first day we will have a panel in which a model family will be created from pre-marital counciling through adolescence of the child. On the program there will be leaders in religion, medicine, nutrition, sanitation, immunization, emotional development of the child, family adjustment, heating and lighting in the home and school, pre-school, teacher and child relationship, exercise and recreation, and general medical care throughout the family cycle. All these subjects will be discussed by leaders in their fields. Following this panel the problems faced in working with exceptional children will be demonstrated by the staff of the Special Education Clinic of Southeastern Louisiana College. This will be followed by a summary of the entire day's program by Dr. F. S. Crockett, Chairman, National Rural Health Committee, A.M.A. On the night of the 28th there will be a banquet with recognition of various celebrities in the field of medicine and health with the dinner speaker being Dr. Charles R. Henry of Little Rock, Arkansas. On Saturday the 29th the program will be a panel discussion on what the rural family and rural physician owe to the community in which they live. This panel will be made up of an outstanding farmer, an outstanding home demonstration expert and the boy and girl winners of the 1955 4-H Club projects. With them will be a physician who knows the obligations of a rural physician. Both programs on the 28th and 29th will be moderated by Mr. Aubrey Gates, National Rural Health Field Representative of the A.M.A.

We believe we have a program that will benefit anyone who will take time to attend this meeting. It is set up on a basis of good will and good public relations. Every doctor and his wife who possibly can should make an effort to attend this meeting. The doctors who are active in the Health Council will be looking forward to your assistance in making this program a success.

M. C. WIGINTON, M. D., President
Louisiana State Health Council

REVENUE BUREAU SUMMARIZES MEDICAL EXPENSES UNDER TAX LAW

Deductible and non-deductible medical expenses for income tax purposes have been summarized by the Bureau of Internal Revenue in a series of rulings that combine new interpretations with a clarification of old rulings. Some examples:

Travel expenses to and from a location where daily visits to a medical clinic are required are deductible but (since 1954) cost of food and lodging are not, except as part of a hospital bill. **On education and training,** special instruction in speech and lip reading for a deaf child are deductible expenses, but not a course of ordinary instruction. Psychiatric care and therapy at specially equipped treatment schools for alleviating mental illness are deductible items, but where cost of instruction at a psychiatric school doesn't represent medical care, it is not deductible. **On health and accident indemnity insurance,** if a policy covers both injury indemnity and medical expense reimbursement, premium cost for latter is deductible but not for former. On **other points,** ordinary exercise rubdown, air conditioner, oxygen equipment, iron lung, special bed board, all are deductible items when prescribed by a physician for an illness, but not food for ulcer patient, maternity clothing, diaper service, wigs or toothpaste.

DIABETES DETECTION DRIVE

This year, as for several years past, the American Diabetes Association is sponsoring a diabetes detection drive for the week of November 13-19. During this week, through the agency of the local medical societies, and with such help from publicity as can be obtained, their detection and education programs in the field of diabetes will be carried out.

The American Diabetes Association is composed of physicians only. Their efforts are dedicated to the furtherance of the general welfare through the acquisition and dissemination of useful and accurate knowledge regarding diabetes mellitus. It undertakes, in the public interest

such activities as will improve the physical welfare of persons having that disorder. Great increase in dissemination of knowledge concerning diabetes has resulted from the activities of this Association.

In the interest of finding hidden diabetics, and in the interest of keeping enterprises that are medical within the profession, it is most desireable that physicians everywhere cooperate in the activities of the Diabetes Detection Drive.

MEDICAL NEWS SECTION

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
Calcasieu	Fourth Tuesday every other month	Lake Charles
East Baton Rouge	Second Tuesday of every month	Baton Rouge
Morehouse	Third Thursday of every month	Bastrop
Natchitoches	Second Tuesday of every month	
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Tangipahoa	Second and fourth Thursdays of every month	Independence
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

WIDER KNOWLEDGE OF CHEMICAL HEALTH HAZARDS NEEDED

The thousands of chemical products developed to make life simpler may only complicate it unless used with care and intelligence.

The Committee on Toxicology of the American Medical Association said recently that there are about a quarter of a million brand name chemical products which may be used in the home, farming, and industry.

All of them may be useful—but handled improperly they may become killers, cripplers, and destroyers of property.

Understanding of the uses and the potential dangers of the wealth of products available is needed to prevent the estimated 3,300 accidental poison deaths which result each year from misuse of chemicals.

The array is so large and so many combinations of chemicals are possible that no complete catalogue of all available products has been made, the committee said.

As part of its campaign to spread information about these products and their hazards, the committee will sponsor a symposium on health hazards of chemicals Dec. 29 during the annual meeting of the American Association for the Advancement of Science, in Atlanta, Ga.

Bernard Conley, Ph. D., secretary of the A.M.A. Committee on Pesticides—which is co-sponsoring the discussion—will be moderator for the symposium. He said the purpose of the meeting is to interpret new knowledge of chemical products to scientists in various fields, so they may in turn use and spread the information.

The committees are working toward development of more intelligent use of chemicals so that their advantages may be enjoyed without dangerous results, Conley said. They do not mean to imply that potentially dangerous chemicals should not be used at all.

"The problem of health hazards has increased with the wider household use of chemicals once found only in industry," he said. "This makes misuse more serious and the necessity for widespread knowledge more urgent.

"There are several thousand basic chemicals used in available products; these can be mixed in an infinite number of combinations and sold under an infinite variety of fanciful names. The products can be changed in composition without notice and even patent office records don't necessarily show the present composition. Thus, a listing of the contents of all brand-name products is impossible to make."

Conley said while no one may know all about all of these products, the danger of poisoning would be greatly reduced by wider understanding of the problem, reasonable care in using any chemical, and careful attention to label instructions.

On the AAAS meeting symposium devoted to the problem in the home, agriculture, and industry will be Lester M. Petrie, M. D., director, preventable diseases service of the Georgia Department of Public Health, Atlanta; Wayland J.

Hayes, M. D., chief of the toxicology section of the Communicable Diseases Center, U. S. Public Health Service, Savannah; Irvin Kerlan, M. D., associate medical director of the federal Food and Drug Administration, Washington; and Mrs. Veronica Conley, assistant secretary of the A.M.A. Committee on Cosmetics, Chicago.

FIND NO MAJOR NEUROLOGIC DAMAGE AFTER 10,098 SPINAL ANESTHESIAS

Results of a study of 8,460 patients given 10,098 spinal anesthetics, one of the largest series to receive "spinals" at one hospital, have revealed no instances of major neurological damage after post-operative follow-ups of as much as five years, it is reported in the **Journal of the American Medical Association** (156:1486, Dec. 18, 1954).

The study was made by Drs. Robert D. Dripps and Leroy D. Vandam at the Hospital of the University of Pennsylvania between 1948 and 1951. Based on their findings and other medical data, they term as "unjustified" the tendency "to assume a cause and effect relationship between spinal anesthesia and a variety of complaints, sometimes appearing years after the anesthesia.

"Our experience indicates that the mortality rate following spinal anesthesia is lower than that recorded after general anesthesia in comparable patients undergoing comparable types of operations."

POISONING TAKES TOLL OF INFANT LIVES

In the United States in 1949-50 the number of deaths from accidental poisoning in children under 5 was 2.6 per cent 100,000 population, or four times that of Great Britain.

One third of them were from drugs—the commonest of which was aspirin.

According to an editorial in the Jan. 22 Journal of the American Medical Association, the problem far exceeds that of leading childhood diseases if you add to accidental deaths the number of children crippled by poisons.

"Anything can be dangerous if taken in excess," the editorial said. "We seem to be a nation of aspirin eaters, and there are few households in which a supply of this drug in one form or another cannot be found." Among the most dangerous to children are sweetened aspirin and aspirin chewing gum.

Other hazards are common household items like, liniment, bleaching agents, lighter fluid, cleaning fluid, insect spray, rat poison, permanent wave solution, shampoo, nail polish remover, antifreeze, detergents, furniture polish, ammonia water, and kerosene.

Because of the problem, a Committee on Toxicology has been established by the A.M.A. to study the health problems of drugs, household goods, and other materials, and to supply educational material to the public. Another important step is the establishment of poison control centers to cooperate with local hospitals.

Most cases of accidental poisoning should be prevented, the editorial said, but when they happen prompt and appropriate treatment can save a life. Cooperative efforts now aiming at solution of the problem "should go far to bring the record of the United States down to that of other highly industrialized countries."

TICK FEVER MAY BE FOUND ANYWHERE

Travel speed and the popularity of western vacations make it possible for Colorado tick fever to show up in any part of the country, U. S. Public Health Service researchers said recently.

In a recent study, virus from the tick which carries the disease was found in patients exposed in California, Colorado, Idaho, Montana, Nevada, Oregon, Utah, Washington, and Wyoming. The disease has been found in persons from as far away as Florida, Maine, and New York.

The study of Colorado tick fever distribution was reported in the Jan. 22 Journal of the American Medical Association by Carl M. Eklund, M. D., Glen M. Kohls, M. S., and James M. Brennan, Ph. D., from the U.S.P.H.S. Laboratory at Hamilton, Mont.

Their isolations of virus were first reported in widespread areas outside of Colorado. They said they found virus-carrying ticks in all nine states where patients had been exposed, and in Canada.

The disease differs from Rocky Mountain spotted fever—also carried by ticks—in that it causes no skin eruption and recovery is invariable. Four or five days after exposure to ticks the patient has chilly sensations, headaches, nausea, and, occasionally vomiting. Fever, headache, and backache last about two days, disappear for about two days, and then return for two or three more days.

DELINQUENCY IS NOT FAULT OF PARENTS OR COMIC BOOKS

Banning comic books and blaming parents is too easy and fruitless a way out of the problem of juvenile delinquency, an editorial in the Jan. 8 Journal of the American Medical Association said recently.

"If we want the delinquent to adjust to society we must recognize the importance of providing the sort of society to which a person may reasonably be expected to adjust," the editorial said.

"The delinquent youth has been described as a rebel without a cause," it said. "He is essentially an unhappy person who is looking for a face-saving way to abandon his false bravado. He does not conform to the standards of a society

from which he feels he has been excluded.

"There is always the danger that the public, goaded by one-sided enthusiasts, will seek an easy but superficial and hence fruitless answer to the problem in the banning of certain types of television programs or 'comic' books. . . . Although in many instances parents are partially to blame for the delinquency of their children, this is too easy an indictment to make, and in almost every case other factors in the child's environment will have been found to play a contributing role.

PUBLIC HEALTH ASSOCIATION PUBLISHES NEW MANUAL ON CHILD HEALTH SUPERVISION

A comprehension professional manual on child health supervision, reflecting present-day emphasis on the inter-relationship of physical and psychological factors, was released in February by the American Public Health Association, 1790 Broadway, New York.

The 180-page volume, "Health Supervision of Young Children", has been five years in preparation, and is the product of collaboration by more than 60 prominent professional persons. It bears the endorsement of the American Academy of Pediatrics, U. S. Children's Bureau, National League for Nursing and Association of State Maternal and Child Health and Crippled Children's Directors.

This is the first of a series of manuals being prepared by the A.P.H.A. Committee on Child Health, under the chairmanship of Dr. Samuel M. Wishik of the University of Pittsburgh Graduate School of Public Health. The text was written by Nina Ridenour, Ph.D., staff consultant to the committee.

Priced at $2, the volume can be ordered directly from the Association.

UNREPORTED CASES OF TUBERCULOSIS

More than 25 per cent of the people who die from tuberculosis come to the attention of health authorities for the first time when the death is reported, according to Dr. Robert J. Anderson, assistant chief, Division of Special Health Services, U. S. Public Health Service, who spoke at the Annual Meeting of the National Tuberculosis Association.

Although deaths from tuberculosis have declined 57 per cent in the past five years, Dr. Anderson pointed out that the decline in newly reported cases has been only about three per cent a year and that at this rate "it will take more than a quarter century to achieve in morbidity the progress achieved in mortality in only five years."

Furthermore, even with progress in finding unknown cases of tuberculosis, he stated, "more than 25 per cent of tuberculosis deaths have never been reported as living cases; many are not reported until admission to the sanatorium; one-third of newly reported cases are far advanced."

The number of active cases in the United States is approximately 400,000, according to Dr. Anderson who said that at any one time about one-third are in hospitals, one-third are patients known to be under treatment at home, and one-third are undetected cases. The number of patients in hospitals, he continued, is larger than at any time prior to 1951 but is slightly less than the peak of two years ago. While there are enough beds for those seeking hospital admission in most areas, Dr. Anderson pointed out there are still shortages in some states.

DISEASE THAT MASQUERADES AS TUBERCULOSIS

A disease that masquerades as tuberculosis but is caused by a newly observed "yellow" bacillus, was reported to the National Tuberculosis Association and its medical section, the American Trudeau Society, recently.

Isolation of a previously unencountered bacillus from 17 patients with a disease which resembled tuberculosis in its clinic manifestations was reported by Dr. Lawrence E. Wood of the University of Kansas School of Medicine, **Kansas City, Kan.** Co-authors of the paper were Dr. Victor B. Buhler, **Kansas City (Mo.)** General Hospital, and Dr. Ann Pollack, University of Kansas School of Medicine. The organism, acid-fast but not typical of this group of organisms, is referred to as the "yellow" bacillus because when grown on artificial medium its color varies from cream to yellow, with a leaning toward orange.

Ten of the 17 patients, according to Dr. Wood, had lesions apparently confined to the lung, with the upper lobe involved primarily. Cavities were frequently evident on X-ray. The age of the patients ranged from a baby one-month old to adults of 65 years. Six of the patients have died, said Dr. Wood, five of them of the unknown disease, with information on the sixth incomplete. The disease is apparently inactive in five patients, who no longer have symptoms, and still another has no X-ray or bacteriological evidence of the disease, but complains of slight cough and tiredness; three patients are still being treated, and two have been lost to follow up, said Dr. Wood.

DISEASE PROCESS PRODUCED BY CALCIFIED LYMPH NODES

"Tombstones" marking the spot of conquered disease within the lung may give rise to a new and more serious disease process.

This strange evolutionary pattern was described recently in a paper presented at a scientific session of the Annual Meeting of the National

Tuberculosis Association and its medical section, the American Trudeau Society, at the Milwaukee Auditorium. The paper was by Drs. Laurence K. Groves and Donald B. Effler of the Department of Thoracic Surgery, Cleveland Clinic Foundation, **Cleveland, Ohio.**

The tombstones are calcified lymph nodes, frequently observed on X-ray near the tracheobronchial tree, which are apparently the residue of healed tuberculosis, histoplasmosis, or some other disease, according to Dr. Groves, who presented the paper. But whatever the cause, the calcified node is the "tombstone" of the original disease, he said, and forms "a mechanical basis for a new disease process," known as broncholithiasis. This disease sometimes simulates cancer.

Broncholithiasis is usually caused when the lymph node erodes into the bronchus, although the primary factor in development of the disease may be a long-standing, active, chronic pulmonary pus formation, according to Dr. Groves.

Symptoms of broncholithiasis include a harassing cough, sometimes accompanied by the spitting up of blood and, in more serious cases, by wheezing respirations when eroding nodes cause bronchial obstruction, he stated. A mild case may be treated by relieving the cough, but surgery may be necessary to remove obstruction in severe cases, said Dr. Groves. He emphasized, however, that the surgical procedure is "extremely difficult and hazardous."

IT MAY BE SCURVY—NOT OLD AGE

Vague, general ill-health wrongly attributed to "old age", may be caused by unrecognized scurvy, T. J. Thomson, M. D., reports from Glasgow, Scotland.* A survey of admission to a general hospital in Glasgow over 15 years revealed 100 cases of florid (bright red) scurvy, but, Dr. Thomson points out, for every case of florid scurvy that comes under medical care, there are almost certainly several minor ones that are never seen by the doctor. Scurvy is preventable, he states; however, some people grow indifferent to the simplest dietetic precautions. Fifty-two out of 58 patients whose records gave details of domestic circumstances lived alone or in lodging houses and did their own cooking.

* **Glasgow Medical Journal, 35**:363 (Dec.) 1954.

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BOOK REVIEWS

Menorrhalgia: Menstrual Distress; by William Drinker, M. D., Springfield, Illinois, Charles C Thomas, 1954, Pp. 97, Price $2.75.

This is a comprehensive monograph divided into eight chapters, clearly written and very easy to read.

The book deals with the various phases of menstrual distress due to anatomic irregularities, disturbed physiology and other imbalances.

It includes chapters on pharmaco-physiodynamics; general management; medical and surgical treatment.

ADOLPH JACOBS, M. D.

Music Therapy; by Edward Podolsky, M. D. (Ed.), New York, Philosophical Library, 1954, Pp. 335, Price $6.00.

This is an anthology of authors from varied backgrounds and experience, all dealing in one way or another with the use of music in various medical situations. The range is from the couch to the electric shock room to the operating room table. The authors have one factor in common: their faith and trust in music as a therapeutic tool. Some of the articles show music in an excellent and immediately practical role in the operating room, as with patients undergoing local or spinal anesthesias; others describe its use in a more remotely useful practice, as in diminishing anxiety in neuropsychiatric states.

Some of the concepts put forth, such as the thalamic reflex, seem undemonstrated and then are used rather indiscriminately to bolster interpretations of the effects of music. One criticism could be levelled at the literal use of the terms ego and id as active agents assessing different aspects of music. There is also a tendency in some of the papers to fall into the error of single cause and effect, emphasizing music as the factor of a psychiatric treatment situation.

All in all, this book attempts an overview of a provocative field, but does it in a way that might tend to discourage the reader from pursuing it further.

IRVIN A. KRAFT, M. D.

PUBLICATIONS RECEIVED

Little, Brown & Company, Boston: Peptic Ulcer, Diagnosis and Treatment, by Clifford J. Barborka, M. D., and E. Clinton Texter, Jr., M. D.

W. B. Saunders Company, Phila.: Counseling in Medical Genetics, by Sheldon C. Reed.

Charles C Thomas, Publisher, Springfield, Ill.: The Postural Complex, by Laurence Jones, M. D.; Differential Diagnosis of Leukoplakia, Leukokeratosis and Cancer in the Mouth, by Ashton L. Welsh, M. D.; Anesthesia in Ophthalmology, by Walter S. Atkinson, M. D.